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PRESIDENTIAL ADDRESS—A REVIEW*

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A PRESIDENTIAL address is difficult to write unless it consists of a scientific presentation and I am going to take but a few minutes of your time in view of the length of the program. This Society, organized in 1917, has progressed so rapidly that I am proud of the honor of presiding at this meeting. In its fold have been gathered practically all of those who, in this country, are placing thoracic surgery upon a scientific and practical basis. Like so many other branches of surgery, development of the art preceded the science and thus in the beginning technical procedures occupied a prominent place in our program whereas today minute studies of the physiology and pathology of disease in the lungs having a bearing upon surgical treatment, occupy the first place.

This year, for the first time, your program committee has been embarrassed by the quantity of material offered and found it difficult to follow the rule of accepting all offerings from members. It seems to me that future committees will find it expedient to select those papers which offer new thoughts or are sufficiently controversial to excite discussion. The plan of devoting one session to a "dry clinic" is most attractive when we meet in cities having sufficient clinical material. Some of the most important subjects which now are attracting the attention of the members of this Society might be cursorily passed in review. The most fruitful source for immediate study is carcinoma of the lung. This disease seems to have been recognized first by Bayle in 1810 and one hun-

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dred years later Adler (1912) stated that it was one of the rarest forms of disease. Since then, however, numerous autopsy statistics have shown that it is as common as carcinoma of the rectum. Unlike the latter disease, its recognition has been slow in developing so that radical attempts at cure have been few and far between. It should be the duty of this Association to preach the need for attention to cough, dyspnea, hemoptysis or pain in the chest in a middle-aged patient and the necessity for bronchoscopy and roentgen ray examination. An exploratory thoracotomy should be undertaken in suspicious cases just as a diagnostic laparotomy is now done in obscure lesions in the abdomen. The operative technic does not offer insuperable difficulties to any expert surgeon. If the patient is prepared by preliminary pneumothorax and phrenic crushing an exploratory thoracotomy is as safe as a laparotomy and no more uncomfortable for the patient, and the surgeon may find a removable lesion. Formerly it was held that only the "central" lung cancers were susceptible of surgical removal but as shown in recent reports bronchial cancers also may be attacked. No doubt technical improvements in time will permit dissection of the regional lymph nodes. The principal difficulty seems to be the failure of the bronchus to heal unless patched over by a fold of lung. But in total pneumectomy this is not possible and we must depend upon a concomitant collapse of the chest by thoracoplasty or else the patient must suffer the bronchial fistula, probably no more than the experience of a colostomy after total removal of the rectum. Perhaps muscle implantation would close a fistula from a larger bronchus as well as it does the smaller peripheral fistulae.

In recent years the treatment of pulmonary tuberculosis apparently has been making rapid strides. For a long time rest was considered the basic principle of treatment but the lung cannot be put at rest by what is called sanatorium treatment. The respiratory rate decreases and the patient's body resistance is strengthened but that is all. If the lung is to be put in a state of rest it must be collapsed and this is the rationale of pneumothorax, phrenicectomy, or thoracoplasty. Abscess cavities in the lung cannot heal except by collapse and fibrous tissue contraction and this ordinarily signifies a shifting of the mediastinum and a flattening of the chest, the former probably leading to a permanent dyspnea. Surgical interference in its simplest form, phrenemphraxis, puts the lung in the position of expiration and recent opinion believes that this relaxation is universal and not basal as formerly held. Elastic recoil is favored. At the present time there is a tendency to favor phrenic interruption over pneumothorax but further balancing of results is needed before the older procedure can be discarded as the method of choice when practicable, particularly as intrapleural pneumolysis is being developed as a fairly easy performance. Certainly the tendency in many

sanatoriums is to increase greatly the scope of collapse therapy particularly in early cases and also not to confine it to unilateral involvement. If these ideas bear fruit thoracoplasty relatively will be infrequently required. We must distinguish between early cases which may need pneumothorax or phrenic crushing and advanced cases which require phrenic exeresis or thoracoplasty. Improvements in technical details will follow a better knowledge of the principles involved. End results, after a five-year apparent cure, will be needed to balance our methods.

Knowledge regarding empyema has reached the point where we should be better acquainted with the exact etiologic factors in its production. The commonly accepted opinion that it is due to the rupture of minute peripheral abscesses may not be the true explanation. Those cases of empyema with bronchial fistula no doubt are secondary to abscess especially in the fissures but otherwise it is probable that studies on lymph flow carrying infection will show that such is more usually the case of the pleural abscess. The treatment of empyema continues to intrigue those surgeons who will not accept the established fact that an abscess must be drained and that the lung will expand and fill the space if drainage is adequate and practiced at the proper moment. The reintroduction of abandoned methods is not common sense.

Abscess of the lung, also, is well settled in clinical practice. We now know that some are embolic in origin and some result from the trapping of a symbiotic group of organisms in obstructed bronchioles. Drainage through the bronchial tubes aided by posture or the bronchoscope will cure many, others must be drained externally. Details of technic which insure adhesion of the pleural surfaces before opening the abscess are being worked out. Some work needs to be done upon the reason for the frequency of cerebral infection.

Bronchiectasis must be recognized in its early stage and prompt measures instituted to cure and keep cured infection in the upper air passages. Only by this can a persistent chronic infection of previous bronchiectatic areas be prevented and the hazards of lobectomy avoided. Once the disease is established all measures are palliative only except lobectomy. It may be that we will learn to recognize a bronchial stenosis following upon respiratory infection as a cause and by manipulation through the bronchoscope prevent atelectatic or bronchiectatic areas from developing. The subject of bronchial stenosis needs further attention.

The tragedy of pulmonary embolus needs further attention. Despite the few brilliant reports of successful operative removal it is probable that in only an occasional case will the staff and equipment be available in time to prevent death. More important are studies which would develop a simple method for determining the clotting power of the blood

used as a routine and preventive measures by diet or other means. A study of the patient before operation for those conditions, such as depressed circulation or lowered metabolism, known to favor stasis of the blood stream, should be part of the estimation of the surgical risk. The older age groups and anterior abdominal wall incisions are important etiologic factors.

Postoperative atelectasis is now sufficiently well known to be recognized easily by clinicians and roentgenologists in practically all cases. A true postoperative pneumonia probably follows atelectasis when the factors of infection and obstruction are present. Some, of course, are embolic in origin. More important is the recognition that hypoventilation (deflation) of the lower lobes follows most cases of operation on the upper abdomen particularly those of the stomach and that early restoration of diaphragmatic descent will lessen the incidence of the more serious complications. The prevention of pain and muscle spasm, avoidance of restricting bandages, deep breathing exercises and carbon dioxide inhalations are all important. Spectacular results will follow bronchoscopic aspiration in cases of massive atelectasis. Some believe that bronchoscopy is indicated early in all varieties of postoperative pulmonary complications.

The etiology of atelectasis is not settled because obstruction by a plug of mucus does not explain cases presumably due to spasm. An acute pleurisy supervening on deflation is an important factor to be considered. Probably some of the unexplained febrile reactions seen after abdominal operations are due to lobular atelectasis with slight infection and which can be detected only by a painstaking physical examination.

Apparently, operative interference in cardiac valve stenosis has not fulfilled expectations. A more promising field has been opened in chronic adhesive pericarditis when there is definite limitation of motion of the heart, thus affecting both its size and function. In addition to the usual well-known reactions, attention has been called to symptoms referable to the gastrointestinal tract, hemorrhage in particular, which may be overlooked or misinterpreted.

We must join with the medical groups in calling attention to the need for operation in certain of these cases. Thoracolysis also may prove helpful in cases of marked cardiac enlargement.

The field of pulmonary surgery has shared with other fields in a destructive attack on the sympathetic nervous system. Particularly in bronchial asthma attempts have been made to relieve the spasm by cutting the vagus or sympathetic filaments reaching the lungs. The operation will suffer the same disrepute that attended sympathectomy for angina pectoris unless complete physiologic and pharmacologic studies are made on each patient in order to determine what should be done. There are limitations to this form of therapy which at the present time

do not justify its use by surgeons generally. We do need a plan either of nerve section or alcohol injection which will relieve pain especially in lung cases similar to that for the relief of the pain of angina pectoris. Carcinoma of the thoracic esophagus is as yet a hopeless disease. Technical methods, so far, have failed to offer any promise. The field has great possibilities by means of the frequency of the disease.

In this short review I have noted only those things which have attracted my own attention in the field of thoracic surgery, some of the problems that have given rise to speculation. This Society is settling most of them and will decide all in the near future, with other problems coming fast for new study.

THE RESULTS OF PHRENIC NERVE OPERATIONS IN 222 CASES WITH A DISCUSSION OF THE TECHNIC OF THE OPERATIONS*

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IT IS the purpose of this paper to report briefly our experience at the Tuberculosis League and Presbyterian Hospitals in Pittsburgh with 222 phrenic nerve operations in a period from July, 1927 to March, 1933; and to record our operative technic. Phrenic avulsion was done 181 times in cases of pulmonary tuberculosis; phrenic crush 22 times. Phrenic avulsion was done 19 times for bronchiectasis.

In the treatment of pulmonary tuberculosis phrenic paralysis has been utilized in moderately and far advanced disease, both unilateral and bilateral, with or without cavitation, and regardless of the location of the lesion in the lung. We have not practiced it for minimal lesions, nor for acute, caseous febrile cases. The primary objective has been to secure a collapse of the lung; an important secondary has been to secure a closure of the cavities. We have used phrenic therapy as a (1) solitary procedure; (2) to supplement artificial pneumothorax; (3) to follow artificial pneumothorax when it is being discontinued, or lost; (4) as a preliminary to thoracoplasty; (5) in an effort to stop massive hemorrhage where artificial pneumothorax is not feasible.

RESULTS

The results in the phrenic avulsion group are recorded in Table I. A little less than one-third (28.7 per cent) of the patients are well and working; a little more than one-third (37 per cent) are improved; and one-third are either unimproved (13.3 per cent), or dead (21 per cent). We have been conservative in our judgment as to the present status of the patients, and have classified our cases accordingly. In no instance do we feel that mortality is to be attributed to the phrenic avulsion. In no case was the phrenic interruption followed by an unfavorable course so closely that it could be ascribed to the operation. Nor in any case have we regretted our choice of permanent avulsion, and wished the corresponding hemidiaphragmatic function restored. In several cases in which there was a later development, or a spread of infection in the contralateral lung, pneumothorax was utilized. Many patients in the improved class, we believe, will proceed to recovery, with or without a supplementary pneumolysis or thoracoplasty. In very few

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TABLE I

PHRENIC AVULSION

	WORKING					IMPROVED					UNIMPROVED					DEAD					TOTAL
YEARS—	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5	
Phrenicectomy alone	1	4	16	4	0	17	7	8	2	0	4	3	7	0	0	16	3	1	1	0	
	25 (26.5%)					34 (35.1%)					14 (14.8%)					21 (22.3%)					94
Phrenicectomy plus artificial pneumothorax	0	8	2	1	1	3	1	6	0	0	3	1	0	1	0	0	1	2	0	0	
	12 (40%)					10 (33.3%)					5 (17.1%)					3 (10%)					30
Phrenicectomy after artificial pneumothorax	0	1	5	0	1	10	1	3	1	2	3					2	1				
	7 (23.3%)					17 (46.7%)					3 (10%)					3 (10%)					30
Phrenicectomy preliminary to thoracoplasty	0	3	0	0	5	2	2	0	0	2					2	5	3				
	8 (33.3%)					6 (25%)					2 (8.4%)					8 (33.3%)					24
Hemorrhage																3					3
	1	16	23	5	7	32	11	17	3	4	10	4	7	1	2	26	8	3	1	0	
Totals	52 (28.7%)					67 (37%)					24 (13.3%)					38 (21%)					181

of those patients classified as well, we think there will be a clinical relapse. In 3 cases of advanced bilateral disease the avulsion was undertaken to stop massive hemorrhage, and was successful in this objective, though all 3 patients died within a few months from progressive disease.

As might be expected a higher percentage recover when phrenic avulsion is combined with artificial pneumothorax, or thoracoplasty; that is, when a maximal collapse of the lung is striven for, and obtained. Again, it is our observation that favorable results are in direct proportion to the rise of the diaphragm. See Table II.

TABLE II

RISE OF DIAPHRAGM

RISE OF DIAPHRAGM	WELL	IMPROVED	UNIMPROVED	DEAD
Two inches or more	25%	52%	14%	9%
One inch	21%	31%	31%	17%
No rise	—	—	57%	43%

This makes it appear that the degree of collapse is a more important factor in healing than the cessation of movement in the diaphragm. Also, as is to be anticipated, success varies indirectly with the amount of the lung areas involved by the disease. See Table III.

In 56 cases where phrenic avulsion was undertaken, alone, with the special objective of closing a sizable cavity, a complete obliteration took place in 13 (23.2 per cent); partial closure in 27 (48.2 per cent); no effect was observed in 16 (26.6 per cent), although 2 of these subse-

TABLE III

PULMONARY INVOLVEMENT

	WELL	IMPROVED	UNIMPROVED	DEAD
Lung Operated Upon (Amount diseased)				
One-third or less	39%	33%	22%	6%
Two-thirds	13%	27%	53%	7%
Entire lung	5%	37%	34%	24%
Contralateral Lung Also Involved (Amount diseased)				
None	47%	33%	13%	7%
One-third	16%	50%	20%	14%
Two-thirds	0	17%	50%	33%
Entire lung	0	0	100%	0

quently became larger. Further analysis shows that sputum was decreased in 70 per cent; sputum became negative in 25 per cent within three months, and in 40 per cent within a year; cough was decreased in 67 per cent of cases. Hemorrhage (cases with a history of recent, solitary, or repeated small hemorrhages) was stopped in 55.5 per cent.

It is often stated that phrenic avulsion used in conjunction with artificial pneumothorax will diminish the incidence of pleural effusion, and will also lessen the need of refills. In our own experience there has been no evidence to support these findings. We are satisfied also that maximal beneficial effect and best results are obtained in lower lobe lesions, although by no means is improvement limited to the basilar lesions.

Phrenic avulsion is often advised as a preliminary measure to test the safety of a planned thoracoplasty. We have not come to place much dependence upon the operation in this rôle. Several times the phrenicectomy gave no clue or warning of a fatal autotuberculinization and an activation of the contralateral lung which followed the thoracoplasty.

Phrenic crush has been done as a substitute for avulsion in a small group of 22 patients. All but 2 had bilateral disease. In 13 the phrenic crush was the only operation. In 5 artificial pneumothorax followed, and in 2 it was given synchronously. Two had avulsion subsequently. In the present status of this group, it is found that 4 (18 per cent) are working; 6 (28 per cent) are improved; 9 (40.5 per cent) are unimproved; and 3 (13.5 per cent) are dead. In one case there was much improvement symptomatically. Diaphragmatic function returned at the end of six months, and with it a relapse. So, a second crush was undertaken. Again progressive improvement was noted in the general condition, cough, sputum, and weight, until a spread of the disease occurred in the opposite lung. In another case of special interest an acute basilar pleurisy, which developed secondary to a fulminating tuberculous peritonitis, subsided immediately following the phrenic crush.

In the light of our experience it would seem that there is a distinct indication for a temporary crushing operation as a trial procedure in (1) extensive bilateral disease; (2) bilateral disease with predominance of the involvement on one side; (3) in a more or less acute, spreading unilateral disease where there is reason to suspect poor resistance, and a likelihood of the contralateral lung becoming involved. A temporary crush can always be made permanent, but the permanent paralysis of the avulsion cannot be undone. Paralysis of the diaphragm will endure at least six months, if the nerve is crushed for 0.5 cm. in length, the amount we have usually chosen, and for a considerably longer period if 0.5 cm. is exceeded. In 6 of our cases paralysis continued beyond a twelve months' period. It is fair to believe that the duration of the paralysis is in direct proportion to the length of the nerve crushed and that if a longer portion than 1 cm. is interrupted, there is a risk of a permanent interference with function.

In our conservatism we have not employed bilateral phrenic therapy. We have felt that there is too much uncertainty and hazard attached to a complete paralysis of the diaphragm. We are using bilateral artificial pneumothorax successfully and safely because the degree of collapse can be quickly and definitely controlled, and made to suit the individual needs. Every now and then there are reports of success following bilateral phrenic paralysis, but these have been of immediate rather than end-results.

Bronchiectasis.—We have done phrenic avulsion in 19 cases of bronchiectasis. This is too small a group of cases upon which to base conclusions of value, and even perhaps impressions, but is included for completeness of record. Six (31.6 per cent) of these patients are potentially well; that is, sputum-free for a period of one or two years; 6 (31.6 per cent) are improved; 7 (36.8 per cent) have not been helped. In 5 of the unimproved group a secondary surgical operation, thoracoplasty or lobectomy, was undertaken before sufficient time had elapsed to allow full phrenic benefit.

It is our impression that it is worth while to try phrenic paralysis before proceeding to serious operations, such as lobectomy or thoracoplasty. If either of these operations is decided upon as a primary procedure, then a phrenic avulsion is needed to help diminish the size of the thoracic cavity. Theoretically, paralysis of the diaphragm diminishes the patient's coughing power, and ability to empty the dependent bronchial tree. Therefore, particularly in those cases in which there is a bronchial stenosis interfering with excretion, phrenic avulsion may not only be ineffective, but even harmful. Under these circumstances one must be prepared for lobectomy or external drainage. However, in practice we have not seen any harm come from a phrenic avulsion. In the majority of our cases, coughing has been made easier and more

effective. We have seen no benefit in other than lower lobe lesions. Prompt and lasting improvement leading to recovery may be expected only in cases of short duration. The longer the life of the bronchiectasis the less likely its exitus by phrenic paralysis.

Operative Technic.—In preparation for the operation, on the night before, the patient is given luminal, gr. 1½; this is repeated two hours

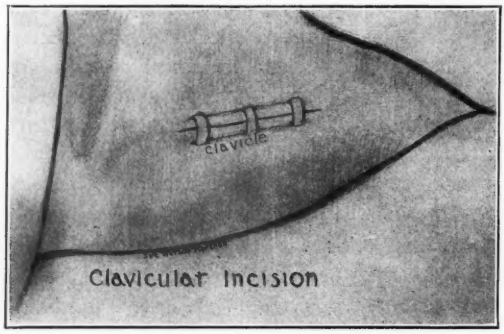

Fig. 1.—Skin incision on the clavicle for phrenic nerve exposure.

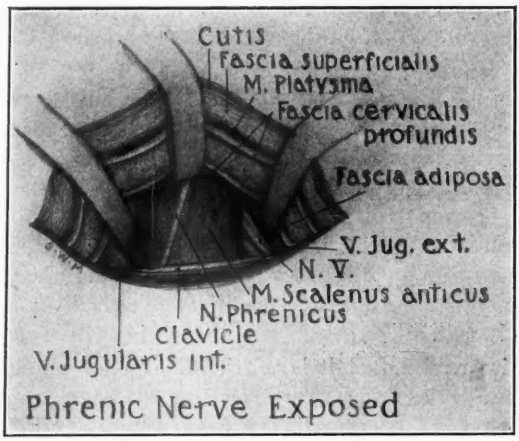

Fig. 2.—Phrenic nerve exposed in subclavian triangle.

before operation, and followed by codeine sulphate, gr. 1, in an hour. In our early experience we administered morphine. A certain number of patients had nausea and vomiting after the operation. Since substituting codeine, gastric upsets have been a rarity. Luminal has served to give a good night's sleep and allay anxiety and nervousness in the most highly strung persons. Many doze through the operation. A folded sheet is placed transversely beneath the scapulae with the patient

supine; the head is turned to the opposite shoulder. Iodine and alcohol are used in the preparation of the skin.

For the exposure of the phrenic nerve, we have used a frontal attack in the subclavian triangle through an incision made on the clavicle opposite the scalenus anterior muscle, which can usually be felt quite easily. We were introduced to this incision by Dr. Lilienthal, who refers to this method of approach in a footnote in his "Thoracic Surgery." We have never used any other incision, nor have we had any desire to change, after observing the technic of exposure made through the cervical incisions at higher levels.

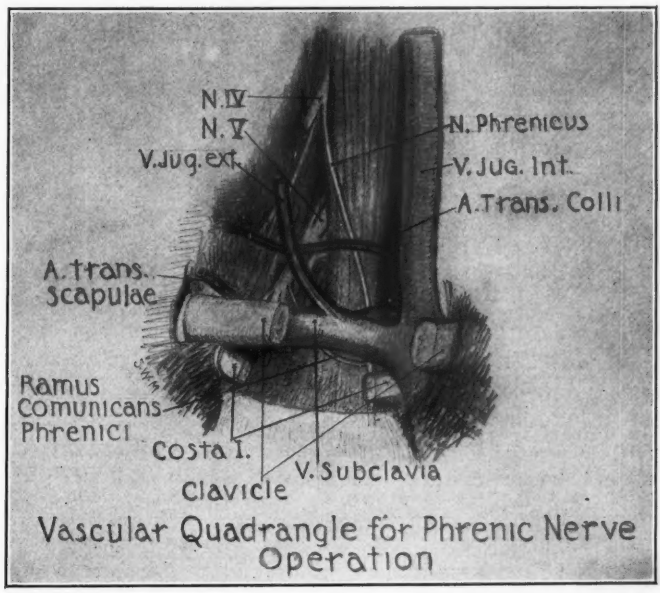

Fig. 3.—Vascular relations in phrenic nerve operation.

The healing and cosmetic effect of the clavicular incision is perfect, due to the rigid support of the clavicle. Rarely is there a conspicuous scar. In at least 50 per cent of our cases it escapes observation except at very close range. The dress covers the area ordinarily. A second advantage is that the phrenic nerve is exposed in a field where accessory phrenics, which eventually join the main trunk below the clavicle, either converge, or are given off from the fifth cervical nerve. This is of particular advantage when a phrenic crush is contemplated, and severance of the accessory phrenics is essential.

Five to 10 c.c. of ½ per cent novocaine solution are injected into the field. An incision 2 to 3 cm. long is made on the clavicle in its long axis through the skin. The subclavian triangle is then opened bluntly

by a Kelly hemostat, close to the clavicle, through the successive layers of the superficial fascia, the platysma myoides muscle, anterior layer of the deep cervical fascia, and finally through a fairly constant fat pad, which may contain a lymph node or two, overlying the scalenus muscle. The phrenic nerve is sought for beneath this adipose layer in a lamella of fascia, on the muscle or along its medial border. This approach is through a quadrangle bounded by blood vessels: internal jugular vein medially, external jugular vein laterally, transverse cervical artery above, and the transverse scapular and subclavian vessels below but behind the clavicle. The jugular veins and the transverse cervical artery may encroach upon the field, and need retraction, as may an unusually broad clavicular attachment of the sternomastoid muscle. For this we have fashioned especially thin, narrow retractors to give adequate exposure without taking up room in the wound that is narrow, and sometimes quite deep. It is rarely that a vessel needs to be ligated. Occasional bleeding from the small veins, which inconstantly cross the field, usually stops itself. Sometimes the ascending cervical vein, which may accompany the phrenic nerve, needs ligation. In only one of our cases has there been a postoperative hematoma, and this was of small size.

When the main trunk of the phrenic nerve is identified, a search is made for accessory phrenic nerves. This is not an essential step, if an avulsion is to be done, but only in the crushing operation, since accessory phrenics, seldom, if ever, join the main trunk lower than the first costal cartilage. A removal therefore of 5 cm. of the phrenic nerve trunk measured from just above the clavicle insures interruption of all the accessory phrenic nerve supply. On this account we are accustomed not to make a tedious search for the accessories. They are divided, if seen.

The main stem of the nerve is injected with novocaine, and cut just below the transverse cervical artery, and a sufficient length twisted out on a hemostat carrying a shoulder to prevent the nerve from slipping. We no longer make a determined effort to secure the whole nerve, or even great lengths, chiefly because this is a painful procedure, and in fact the only painful part of the operation. Other potential complications, such as tearing into the pleura or lung, rupture of the pericardiacophrenic vessels, injury to the diaphragmatic sympathetic nerve communications are avoided. These sympathetic communications are, in general, minute, but go directly to the semilunar ganglia, and theoretically might be a factor in producing shock. As a final step three Michel's clips are placed in the skin, and a small dressing fastened snugly in place with adhesive plaster. Our average operating time is ten minutes. In a few cases there has been difficulty in locating the phrenic trunk. Then, it has been found to be ectopically under cover of the medial or lateral borders of the scalenus anterior muscle, and in one instance was buried by muscle fibers.

Complete paralysis was secured in all but two cases. In one of these, even though the patient complained of characteristic pain in the neck, shoulder, and thorax as traction was made, we probably mistook a small laterally placed vagus for the phrenic. Hoarseness of the voice, elevation of pulse (five to ten beats), and a tendency to flatulence followed. This woman, within several months, developed symptoms of dementia for which condition she was transferred home, and we have been unable to find her for follow-up record. In another case only a partial paralysis

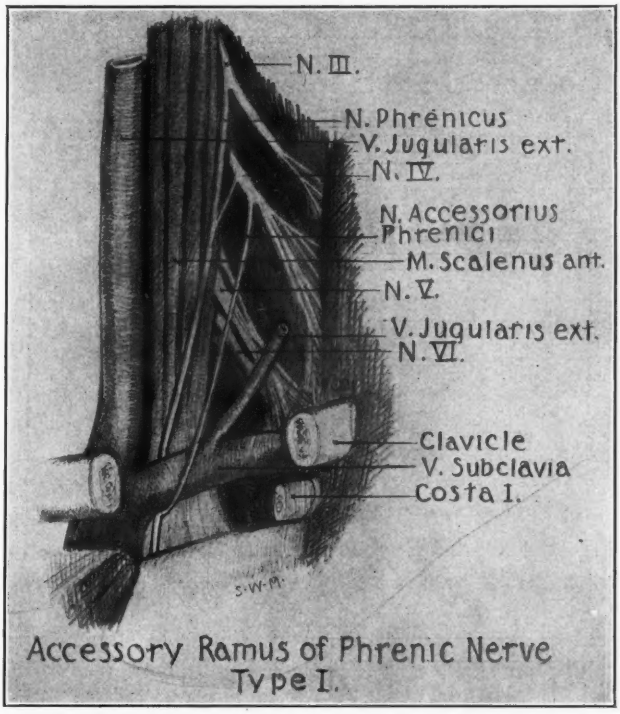

Fig. 4.—Type I accessory ramus of phrenic nerve given off from the third or fourth cervical nerves—vertical course.

of the diaphragm was obtained. The central portion seemed inactive, but the lateral peripheral portion functioned normally. A second operation was done, and then a sizeable nerve, probably an accessory, was found on the scalenus medius muscle, overlapped by the scalenus anterior. Now, if there is any question of identification, or of success in securing the paralysis, the patient is wheeled to the fluoroscopic room, close to the operating room, and checked up before closure of the incision is made.

One other nerve complication occurred in our series. This patient developed Horner's syndrome, which disappeared completely in three months' time. It was supposed in this case that the sympathetic cervical trunk had in some way been traumatized. Other surgeons have had the same experience. The accident raises the question of the connecting rami between the phrenic and cervical sympathetic trunks. We have found occasionally on the cadaver, quite a sizeable branch between the two lying above the level of the transverse cervical artery. It seems reasonable, though perhaps speculative, that in twisting out the phrenic, occasionally a stout connecting ramus holds sufficiently to lacerate, or

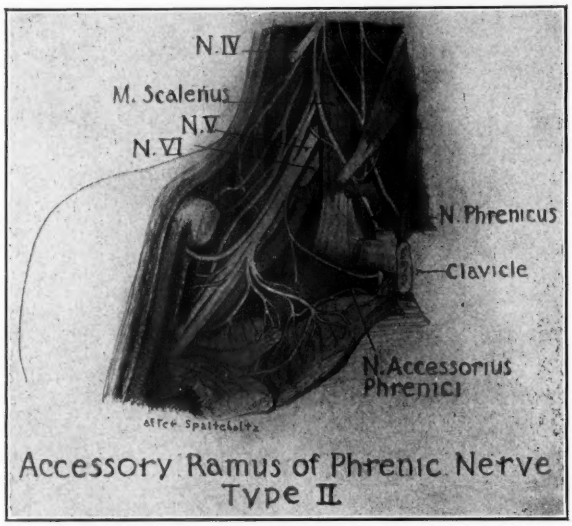

Fig. 5.—Type II accessory ramus of phrenic nerve from fifth cervical nerve—horizontal course.

injure the sympathetic trunk. At least in this explanation one finds some measure of comfort when he feels that he did not traumatize the trunk directly.

There has been in published reports considerable discrepancy about the frequency and the location of the so-called accessory phrenic roots and rami; the occurrence being cited as high as from 70 to 80 per cent by Goetze and Alexander, while Matson and Plenk and Felix record as low as from 20 to 30 per cent. We suspect that the variance is due in part to the lack of definition as to what constitutes an accessory root. It is entirely normal and usual for the phrenic trunk in its formation to have roots from the third, fourth, and fifth cervical nerves. These are short, and blend immediately with the main trunk, and may be properly called roots. Occasionally, they may be found coming from the second and sixth cervical nerves.

Accessory phrenic nerves, on the other hand, while derived from the same cervical nerves, or branches of them, are long and join with the phrenic trunk close to the clavicle; and about 75 per cent of the time below it, in the vicinity of the first costochondral joint. In our own dissections of 72 nerves on the cadaver we have found accessory phrenics in 30 per cent. At operation we have found them 35 times (15 per cent), but undoubtedly have overlooked many. Accessory phrenics are of two general types:

1. A branch derived from the third or fourth cervical nerves or cervical plexus, pursuing a vertical course downward on the scalenus anterior muscle, or lateral to it, and joining the phrenic trunk just above the clavicle, or near the first costochondral articulation. It is this arrangement which gives rise to the appearance and nomenclature of a bifid nerve.

2. In a more frequent group, a branch is given off from the fifth cervical nerve, or from the nerve to the subclavius muscle (in turn derived from the fifth), or from the sixth cervical, and has a short, vertical course, lateral to the scalenus anterior muscle, and then swings horizontally across the muscle at the level of the clavicle, to join the phrenic trunk at or near the first costal cartilage.

The accessory nerves on the cadaver dissections are tabulated. According to this classification (Table IV) they differ but little from those recorded by other observers. We did not find more than one accessory in one field. The unusual finding was one given off from the nervus cutaneous colli. The common finding was a branch from the nerve to the subclavius.

TABLE IV

NUMBER	ORIGIN	POINT OF JUNCTION WITH MAIN TRUNK
		Group 1
1	C III	1 cm. above clavicle
2	C IV	3 cm. above clavicle
3	C V	Costal cartilage 1
4-6	Junction C IV-V	Costal cartilage 1
7	C IV	(Descended on scalenus medius) costal cartilage 1
8	N. cutaneous colli	Above clavicle.
		Group 2
9-10	C V	1.5 cm. above clavicle
11	C V	Behind clavicle
12-13	C V	Costal cartilage 1
14-22	Nerve to subclavius muscle	Costal cartilage 1

The accessory nerves as they join the main phrenic trunk below the clavicle, lie either behind, or in front of the subclavian vein, and if in front, form a loop which tends to exert pressure against the vein when traction is made upon the main stem in the process of avulsion. Our

observation is that the accessory filaments are so slender that they will break before any injury or tearing of the vein wall can take place, so that the danger from this source is infinitesimal. While deaths following hemorrhage have been reported, we know of none definitely from subclavian vein injury. Injury to the pericardiacophrenic vessels, thyrocervical trunk, and internal jugular vein have been recorded.

Postoperatively our patients have had an uneventful convalescence. There has been no wound infection; and no evidence of injury to the thoracic duct. We have been fortunate in that there have been no unfavorable sequelae, such as pneumonia, atelectasis, embolism, and immediate spread of tuberculous infection; nor has there been any evidence of autotuberculinization, which has manifested itself several times following thoracoplasty. One patient, in whom a four-inch rise of the diaphragm had been secured on the left side, had at times severe palpitation and flatulence. These symptoms became so annoying and persistent, that an artificial pneumothorax was induced, and complete relief obtained.

In conclusion, on the basis of our comparatively limited experience, we can testify to the great value and the safety of phrenic therapy in the treatment of pulmonary tuberculosis, and to a less degree of bronchiectasis. There seems to be need for a more precise knowledge and understanding of the indications in treating bilateral disease. We probably have been overconservative in the selection of cases. We have, however, been working in a virgin field, in which our patients, rather than demanding operative procedure, have had to be persuaded to it. We have been fortunate in not having cause to regret our phrenic operations thus far, but we realize the fickleness and errancy of tuberculous infection; how overnight a perfectly rosy picture can, without rhyme or reason, be darkened by a bursting spread in a new lung field. Therefore, the best laid plans and procedure will go astray, and phrenic therapy must at times be wrongly chosen, and at other times fail to secure favorable results. In the vast majority of cases, however, it will alone, or in combination with other methods, contribute to the success of the objectives of collapse therapy; hasten recovery, save economic loss, and restore economic independence. Its value as a public health measure in limiting the spread of the infection by reducing sputum and the incidence of tubercle bacilli in the sputum, cannot be overestimated.

As for the operation itself, we think that it is a relatively simple one, but that it requires exact and careful technic. It is performed in an anatomic field fraught with sufficient potential danger to demand the skilled and experienced, rather than the occasional operator.

AN ESTIMATE OF THE VALUE OF PHRENIC NERVE INTERRUPTION FOR PHTHISIS BASED ON 654 CASES*

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EXTREMES of published opinion as to the value or uselessness of hemidiaphragmatic paralysis for pulmonary tuberculosis continue to appear to the confusion of all who are anxious for the balanced truth. Some reported series of cases showing almost phenomenally good results largely represent patients whose tuberculosis was diagnosed relatively early at well-organized city clinics and who were treated by phrenic nerve interruption rather than pneumothorax almost as soon as they were admitted to a sanatorium. Some of the excellent results attributed to the phrenic operation would undoubtedly have been obtained from unaided bed rest. Parenthetically, we do not intend to imply that phrenic operations had been injudiciously applied in these cases. A quite different situation exists in many clinics in health resorts which receive relatively few patients with early tuberculosis, but many with chronic fibrotic far advanced cavernous lesions. In this group, phrenic operations are likely to be disappointing with respect to aiding in complete closure of pulmonary cavities and arrest of the disease. Obviously, the expertness with which a phrenic operation is performed has, in contradistinction to thoracoplastic operations, nothing to do with its effect upon the tuberculosis, provided that no accident occurs and that a complete paralysis is produced.

The detailed statistical study that we are presenting fairly represents, we believe, a cross-section of that part of the population that is under medical treatment for active pulmonary tuberculosis. Because our patients have been drawn from many different sources, neither of the groups mentioned in the last paragraph is disproportionately represented. In contrast with many of the patients whose results have been reported from abroad, the great majority of our patients have been financially able, usually through public assistance, to persist in the proper postoperative care of their tuberculosis.

It must be fully recognized that phrenic paralysis per se cannot be given the whole credit or blame for the good or bad clinical results that are reported in any series of cases. Not a few of the good results that we are reporting would undoubtedly have been obtained from the

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sanatorium regimen, unaided by phrenic paralysis. There is, therefore, no intended implication in our presentation that the phrenic operations that were used in this series of cases in conjunction with the sanatorium regimen, were necessarily solely responsible for the good results that occurred. We do believe, however, that the results that we are reporting are far superior to the results that could have been expected on the basis of common experience in patients with identical lesions if bed rest alone had been used. As we are convinced from our own and others' experiences that phrenic paralysis increases the likelihood of healing and hastens it, we use the operation in suitable cases rather than entrust these patients to the less effective therapy of bed rest alone. An estimate of the true value of phrenic paralysis, as also of bed rest, should obviously be based upon results in large series of cases rather than upon a few striking instances of remarkable success or dismal failure.

The 654 patients who have had some operation on the phrenic nerve for pulmonary tuberculosis during the six-year period ending in the autumn of 1932 are represented in our study. One hundred twenty-eight patients have had phrenic operations for pulmonary abscess or bronchiectasis, three for diaphragmatic hernia, two for pulmonary holes remaining after removal of echinococcus cysts and three for miscellaneous indications, are not included in any of our figures, nor are approximately 150 additional operations performed for tuberculosis and other diseases during the past six months.

We have been unusually fortunate in having been able to trace by recent personal examination, report from the present attending physicians or from the patients' reply to a questionnaire, 612, or 93.5 per cent of the 654 patients. As the labor involved in assembling the data that are the subject of this article occupied absolutely the entire time of one of us (L. W. N.) for eight months, it is not likely that such studies as this will be frequently repeated.

Fifty-eight and nine-tenths per cent of the patients were female and 41.1 per cent male. The patients ranged in age from two to sixty-three years. Fifty-three and four-tenths per cent of the operations were left-sided and 46.6 per cent right-sided. Of the left-sided cases, 32 per cent became arrested, 35 per cent were improved, 13 per cent remained stationary, 6 per cent became worse and 13 per cent have died. Of the right-sided cases, the corresponding figures are: 35 per cent, 35 per cent, 10 per cent, 4 per cent, and 15 per cent. There is, therefore, virtually no difference between the results in the left-sided and right-sided cases.

Three hundred two patients had no other collapse therapy measure than phrenic nerve operation. One hundred eighty-eight had a phrenic operation secondary to an inadequate pneumothorax. In 66 patients

phrenic operation proved to be insufficiently effective and pneumothorax or further surgery became necessary. This group does not include the following 67 cases in which the phrenic operation was performed without the intention of waiting for a completely successful clinical result from it alone, but in which some complementary operation was expected to follow it within a few weeks: Phrenic operation was performed in conjunction with thoracoplasty in 51 cases, with scaleniectomy in nine, with multiple intercostal neurectomy in four and with extrapleural pneumonolysis in four. It may be said parenthetically that the number of cases of thoracoplasty, et cetera, given here does not represent the total number of these operations used in the 654 cases that are the subject of this article, for the reason they were also used in the 66 cases in which an initial phrenic paralysis proved to be clinically unsatisfactory and a few of these operations were finally used when a pneumothorax-phrenic sequence was found to be clinically unsatisfactory.

In 25 cases a phrenic operation was performed on the side opposite to an existing induced pneumothorax. Miscellaneous cases are three in which phrenic operation was used bilaterally, but only after diaphragmatic motion on the first side had returned after a temporary phrenic nerve interruption, and three in which phrenic operation was used for the childhood type of tuberculosis.

A detailed consideration of our indications and contraindications for phrenic nerve surgery is beyond the scope of this study. Results, however, will be reported with reference to such factors as the extent of the lesion, the severity of the symptoms, the presence or absence of tubercle bacilli and of a pulmonary cavity, the size and position of the cavity, the age of the patient and of the lesion, the influence of operation upon fever, weight, cough, hemoptysis and amount of sputum.

We have undertaken to estimate whether or not paralysis of the hemidiaphragm has fulfilled our expectation of it, which varied from complete healing of the lesions to mere symptomatic relief of some distressing symptom. Among the total of 652 patients (two from the grand total of 654 are excluded from all estimations of the value of phrenic paralysis because operation completely failed to paralyze the diaphragm; both of these were in patients for whom the operation was intended as the sole procedure) the expectation was fulfilled in 58 per cent* and was not fulfilled in 30 per cent. More than 70 per cent of the operations were performed from six months to five and a half years before the time of estimation of the result.

*Throughout this article we have eliminated fractions of a per cent and have used the whole figure that is closest to the actual true figure. Frequently, therefore, the percentages given will not total exactly 100 per cent. We believe that elimination of distracting fractions will assist the reader to a somewhat clearer appreciation of the values that we present.

TECHNICAL OPERATIVE CONSIDERATIONS

As to the type of operations performed, 66 per cent were permanent paralyses, 33 per cent temporary and 1 per cent questionable because of lost operative notes; 20 per cent of the temporary interruptions were later converted into permanent interruptions.

Except for a few operations performed in 1920, all of our early permanent paralysis operations were evulsions or exereses of the phrenic nerve. Although it happens that we had no operative accident from blindly tearing the nerve from the mediastinum, we disliked to do it and we have become increasingly impressed with the number of fatal or grave complications that have been reported as being directly due to the evulsion itself; for these reasons and because evulsion under local anesthesia always causes pain, which is often severe unless a general anesthetic is used, we abandoned the operation except in those cases in which we felt in doubt as to finding all accessory phrenic roots in the Goetze operation, which we now greatly prefer. Forty per cent of our permanent paralysis operations have been of the Felix exeresis type and 60 per cent of the Goetze type; the latter figure will constantly increase in our clinic.

Neither type of operation assures complete paralysis of the hemidiaphragm. If in the Felix operation the smaller of two or more phrenic roots is the first found and the only one looked for, its evulsion may tear it loose from the larger phrenic trunk which therefore remains functionally intact; also, if the largest root be chosen for evulsion and if the nerve ruptures short of the insertion of lesser roots into it, a lesser root is likely gradually to assume full motor function. Many reported series of cases cite failures to obtain paralysis after evulsion; by mere chance it happens that this did not occur in any of our 179 cases. In the Goetze operation, failure to find and resect 2 or 3 cm. of all of the motor roots of the phrenic nerve is equally likely to result in incomplete paralysis. This occurred in 11 of our 257 cases. Eight of these 11 were operated on by junior surgeons in our clinic. Five of the 11 were promptly reoperated on and the diaphragm became completely paralyzed. Two others were reoperated on; in one the initial paralysis of approximately two-thirds of the diaphragm was not improved upon and in the other no paralysis whatever was effected. It is possible that an anastomosis, which anatomic dissections occasionally show to be present between the phrenic and sympathetic nerves below the level of the clavicle, may be responsible for continuing diaphragmatic motion after all the roots of the phrenic nerve itself have been resected. Four were not reoperated on because there had been complete paralysis for five, seven, seven, and nine months, respectively, and the disease had become arrested in one case, quiescent

in two (following a subsequently induced pneumothorax in one of them), and improved in one. The principal cause of failure to produce complete paralysis at the first operation was the mistaking of one large or several small accessory phrenic roots for the main stem; in two cases the cause of initial failure was mistaking the sympathetic for the main phrenic stem and in one case the main phrenic was found at the repeated operation to be within the belly of the scalenus anticus muscle. In two of the three initial operations (which were performed early in his experience with the Goetze technic) at which the senior surgeon failed to produce complete paralysis, because of having divided only accessory roots, he later found the main stem far mesial under the carotid artery and in the third case paralysis was complete for nine months, at which time the tuberculosis was arrested and reoperation was not indicated. Three of 217 temporary phrenic paralyses were not made complete at the first operation; this may be an underestimation of failure at the initial operation because not all patients were fluoroscoped after operation. Paralysis was made complete at reoperation in two of the three and the third was not reoperated on.

In those cases in which we have used the Felix operation, the length of the evulsed section of nerve has been measured in 184. Three per cent were 5 cm. or less in length; 20 per cent, 6 to 10 cm.; 18 per cent, 11 to 15 cm.; 11 per cent, 16 to 20 cm.; 14 per cent, 21 to 25 cm.; 20 per cent, 26 to 30 cm.; 9 per cent, 31 to 35 cm.; 3 per cent, 36 to 49 cm. Forty-four, or 23 per cent, of these 184 patients had less than 11 cm. of the phrenic nerve evulsed and were therefore subject to continuing motor function through an accessory phrenic root which, it has been found, usually inserts into the main phrenic stem from 10 to 12 cm. from the usual site of division of the main stem in the cervical operative incision. The longest nerves that were removed in our series were 49 cm., 40 cm., 37.5 cm., and 37.5 cm.

We have repeatedly seen patients with bilateral tuberculosis in whom was demonstrated the tragedy of a permanent paralysis having been produced. The operation may have brought about arrest of the lesions in the lung of the operated side but what reduction in total respiratory function the paralysis caused has not rarely prevented our performing some effective operation on the opposite side for lesions that may later have progressed there. For this reason we have rarely during the past two or three years induced a permanent paralysis in the presence of bilateral active lesions until we have observed whether or not the four, five, or six months of paralysis that is obtained from a temporary operation (phrenemphraxis) is sufficiently satisfactory to justify making the paralysis permanent, or perhaps repeating the temporary paralysis. Twenty per cent of all of our temporary paralyses were later converted into permanent ones.

After return of diaphragmatic motion after temporary paralysis, four patients suffered a reactivation of their lesions; reopening or enlargement of their cavities occurred in three of them. In three of the four a permanent paralysis was then induced and all regained their former improvement. The fourth patient did not return for reoperation.

COMBINED PROCEDURES FOR BILATERAL TUBERCULOSIS

In three patients temporary phrenic nerve interruption (in one case in conjunction with pneumothorax and intrapleural pneumonolysis and in one with pneumothorax alone) was successful in controlling the lesions on the operated side, but lesions that had been present in the opposite lung at the time of operation continued to progress. For these lesions the phrenic nerve was interrupted in preference to attempting to induce pneumothorax, but only after diaphragmatic movement returned on the side of the original phrenic operation. The tuberculosis in one of these patients is arrested, in one quiescent and in one improved. We have never performed even a temporary bilateral phrenic interruption for pulmonary tuberculosis and we are strongly opposed to a bilateral permanent interruption.

In 25 patients pneumothorax was used on one side and phrenic nerve interruption on the other side. In 17, pneumothorax and in eight, phrenic operation was used on the first side; in 11 of the 17, phrenic operation was the first choice for the second side and in six, pneumothorax was, but induction being impossible, phrenic operation was performed. Eighty-four per cent of the 25 phrenic interruptions were temporary and 16 per cent permanent. Seventy-six per cent of the patients were improved and 24 per cent unimproved, worse or dead. The 25 patients may be divided into four groups: 1. Ten patients in whom the phrenic operation was used to close a cavity that had developed in the lung opposite to that collapsed by pneumothorax. This was successful in eight, whereas the cavity enlarged in two, and one of these later died. 2. Eight patients in whom the operation was similarly used to control a noncavernous lesion. In four of these the lesion has cleared with corresponding clinical improvement. In three patients (two of whom had had "spreads" following induction of pneumothorax) the lesions have apparently remained stationary and in another patient they progressed and the patient died. 3. In two patients the operation was used primarily to stop hemoptyses that were presumably coming from the side opposite to that of a successful pneumothorax. In one the hemoptyses stopped and general improvement followed; in the other the severe hemorrhages continued and the patient died four days after operation. 4. Five patients in whom a phrenic operation was successfully used in preference to a pneumothorax for the lesions of the originally worse

lung, later developed progressive lesions in the opposite lung and for these a pneumothorax was induced. This was technically and clinically successful in four of the five cases.

OPERATIVE AND POSTOPERATIVE COMPLICATIONS

On several occasions we have had some bleeding from injury to the venous network that is frequently present in the field of a phrenic operation. In all cases except one, this complication was trivial and the bleeding was easily controlled by gauze pressure or by ligation. In one case a vein, which was not visible at the time it was injured, continued to bleed in spite of prolonged pressure, and attempts to place a hemostat upon the bleeding point increased the size of the opening in the vein, which later was found to be the internal jugular near to the angulus venosis. Hemorrhage was then completely controlled by holding the tip of a finger in the rent in the vein while a pursestring suture was passed around the rent which was thereby firmly closed. Preliminary ligation of the vein above the rent had no apparent influence on the bleeding, and ligation below the rent was impossible because of its proximity to the angulus venosis. The phrenic operation was completed and the patient made an uneventful recovery, not having lost enough blood to affect him adversely. Similar accidents have been reported, some being fatal from air embolism, and I have heard of one patient who died within a few hours of operation from a tremendous venous hemorrhage that could be controlled only after the clavicle was divided.

Horner's syndrome of cervical sympathetic paralysis is an occasional operative complication and it usually disappears within a few weeks. In three of our cases, however, it persisted. Temporary paralysis is probably due either to the local anesthetic or to slight traumatism by the mesial retractor or other instrument. Permanent paralysis was probably due in our three cases to the sympathetic trunk having been mistaken for the phrenic nerve by junior surgeons, although the possibility of its being caused by division of the occasional nerve that connects the main phrenic and the sympathetic trunks must be considered. This mistake need never occur if every nerve that is believed to be a phrenic root be traced upward, enlargement of the incision being unnecessary, in order to determine whether or not it comes from the cervical roots of the brachial plexus. In one patient the long thoracic nerve of Bell was resected by mistake for the phrenic at the first phrenic operation performed by a junior surgeon.

Wound infection, which was not severe, occurred in only two patients.

Slight postoperative dyspnea has been noted in 52 cases, although the majority of patients are aware of some dyspnea on exertion, which may in part be due to pulmonary fibrosis or to the temporary reduction

in cardiorespiratory efficiency that occurs in some fat patients who have long been confined to bed. In only one patient was dyspnea severe. It immediately followed operation, which caused a 4.5 cm. rise of the left diaphragm, and it has persisted during the year and a half that has passed since operation, though it has been much relieved since induction of pneumoperitoneum. The preoperative lesion had been a predominantly unilateral far advanced cavernous productive one of five years' duration in a forty-four-year-old man with extensive fibrosis throughout both lungs; a vital capacity reading had not been taken. During the eighteen months since operation the patient has become worse and the cavities have increased in size. This is the only patient among the 654 whom we consider to have been permanently harmed by phrenic paralysis, except those whom we have already mentioned as having been prevented from having some effective collapse therapy measure performed for newly progressive lesions on the side opposite to that in which a permanent, rather than a temporary, phrenic paralysis had originally been performed.

Hemoptysis complicating phrenic operations will be considered when the effect of phrenic paralysis upon hemoptysis is detailed.

In only one case is it probable that a phrenic operation was directly responsible for an extension of the disease in the slightly diseased opposite lung. In this case a Goetze operation was followed on the next day by a pneumonic type of infiltration in the lower half of the opposite lung where some active tuberculosis had been present before operation; the fresh lesions were successfully controlled by an induced pneumothorax. In addition to this case tuberculous lesions of the progressive type, which were present in the opposite lung before operation continued to progress, but not precipitously, after operation in 15 cases among the 272 cases in which phrenic paralysis was used independently, but we feel that progress was not hastened by the operation.

Gastrointestinal symptoms worthy of note occurred in four patients. Three of them had had left-sided phrenic operations. In one, whose diaphragm rose 3 cm., there were nausea and vomiting for two weeks; in another, whose diaphragm rose 5 cm., there were "nausea and vomiting which were relieved by frequent small feedings" and in the third, whose diaphragm rose 3.5 cm., nausea and vomiting have occasionally occurred during the two postoperative years. This rather neurotic patient in whom symptoms of nausea, loss of appetite and a 19-pound loss of weight followed a right-sided phrenic operation performed two years ago is apparently cured, and although his diaphragm is absolutely paralyzed, it has not risen any and it is pulled down considerably in inspiration by the unparalyzed half of the diaphragm.

The amount of lateral displacement of the heart following phrenic paralysis has been measured in the 74 patients in whom the diaphragm

has risen 5 cm. or more and in whom the phrenic operation was used alone. The average shift of the heart to the right after the 37 left phrenic paralyses was 0.93 cm. and to the left after the 37 right phrenic paralyses was 1.33 cm. In 46 per cent of the left and in 8 per

TABLE I

RESULTS ACCORDING TO LENGTH OF POSTOPERATIVE OBSERVATION

MONTHS	CASES	ARRESTED	IMPROVED	STATIONARY	WORSE	DEAD
		%	%	%	%	%
3-12	117	15	52	13	4	14
12-24	80	40	27	11	5	16
24-36	59	57	17	11	6	6
36-72	12	66	25	8	0	0
Unknown	4	0	0	0	0	100
Total	272	34	35	12	5	14

As no patient may be listed as *Arrested* until he has had no signs of active tuberculosis for at least six months, a number of cases now listed as *Improved* will soon automatically enter the *Arrested* group.

The criteria as to the status of a tuberculous person, as defined by the National Tuberculosis Association in 1931, have been used by us in classifying our results, with the exception that we have substituted *Stationary* and *Worse* categories for the Association's *Unimproved*. Furthermore, we have combined its *Apparently Cured* and *Arrested* categories under the single heading of *Arrested*, and its *Apparently Arrested*, *Quiescent* and *Improved* categories under *Improved* for the reason that partition of our cases into few rather than many groups gives a clearer conception of the value of phrenic paralysis. The following are our abbreviations of the N. T. A.'s definitions of the various categories: 1. *Apparently Cured*—No constitutional symptoms, tubercle bacilli nor roentgenologic evidence of active lesion for at least two years under ordinary conditions of life. 2. *Arrested*—Essentially the same as *Apparently Cured* except that these conditions must have been present for at least six months, during the last two of which the patient has walked for one hour twice daily or taken equivalent exercise. 3. *Apparently Arrested*—Exactly the same as *Arrested* except that the minimum time is three rather than six months. 4. *Quiescent*—No constitutional symptoms; tubercle bacilli absent or present; roentgenologic evidence of stationary or retrogressive lesion. These conditions must have been present for at least two months, during the last month of which the patient has exercised for thirty minutes twice a day. 5. *Improved*—Few or no constitutional symptoms; tubercle bacilli present or absent; roentgenologic evidence of stationary or retrogressive lesion.

TABLE II

RESULTS ACCORDING TO NATIONAL TUBERCULOSIS ASSOCIATION CLASSIFICATION OF LESIONS AND SYMPTOMS

N. T. A. CLASSIFICATION	CASES	ARRESTED	IMPROVED	STATIONARY	WORSE	DEAD	ABILITY TO WORK	
							TOTAL	PARTIAL
		%	%	%	%	%	%	%
Minimal	39	69	20	10	0	0	41	20
Moderately Advanced A	34	41	50	6	3	0	20	11
Moderately Advanced B	82	38	46	5	1	10	17	17
Far Advanced 1	21	38	28	9	9	14	0	33
Far Advanced 2	86	14	31	24	9	19	2	15
Far Advanced 3	10	0	0	0	0	100	0	0
Total	272	34	35	12	5	14	11	18

cent of the right phrenics there was no shift. Among the left phrenics there was a shift of less than 2 cm. in 39 per cent and between 2 and 3.6 cm. in 13 per cent; in the right phrenics the corresponding figures are 75 per cent and 16 per cent. In no case did the shift exceed 3.6 cm. There is no evidence that the shift was manifested by symptoms in any case.

TABLE III

INDICATIONS, TYPE OF OPERATION, AND TUBERCLE BACILLI

N. T. A. CLASSIFICATION	CASES	BROAD INDICATIONS			TEMPORARY OR PERMANENT PARALYSIS			TUBERCLE BACILLI*			
		CLEARLY FIRST CHOICE	SLIGHT PREFERENCE TO PN'THORAX	AFTER TRYING PN'THORAX (IMPOSSIBLE)	TEMPORARY	TEMPORARY MADE PERMANENT	PERMANENT	SPUTUM BECAME −	SPUTUM STILL +	SPUTUM − BEFORE AND − NOW	SPUTUM − BEFORE AND + NOW
		%	%	%	%	%	%	%	%	%	%
Minimal	39	84	3	13	38	26	61	2	0.6	86	3
Moderately Advanced A	34	89	9	2	44	33	56	5	1	67	0
Moderately Advanced B	82	70	17	12	36	36	63	21	5	50	0
Far Advanced 1	21	62	0	38	48	50	52	5	3	35	0
Far Advanced 2	86	36	8	56	37	2	63	16	32	10	1
Far Advanced 3	10	70	0	30	20	0	80	0	7	0	0
Total	272	63	9	27	38	28	61	50	50	41	0.8

*Percentages in first two columns based on the 149 patients whose sputum was positive before operation.
Percentages in last two columns based on the 259 patients whose records are complete as to tubercle bacilli.

RESULTS FROM PHRENIC PARALYSIS USED INDEPENDENTLY

Some type of phrenic operation has been used independently without the preliminary or subsequent use of pneumothorax or other collapse therapy measure in 302 patients, 272 of whom have been traced.

TABLE IV

RESULTS ACCORDING TO AGE OF PATIENT

AGE	CASES	ARRESTED OR IMPROVED	STATIONARY, WORSE OR DEAD
		%	%
5-10	1	100	0
11-15	9	77	23
16-20	40	80	20
21-25	53	77	23
26-30	63	76	24
31-35	28	60	39
36-40	12	33	66
41-45	20	55	45
46-50	17	70	30
51-55	13	39	61
56-60	3	66	33
60+	3	33	66
Unknown	10	70	30
Total	272	69	31

TABLE V

RESULTS ACCORDING TO AGE OF LESION

AGE OF LESION (MONTHS)	CASES	ARRESTED OR IMPROVED	STATIONARY, WORSE OR DEAD
		%	%
1- 3	12	91	9
4- 6	27	90	10
7- 9	31	68	32
10-12	23	53	47
13-15	14	72	28
16-18	21	77	23
19-21	13	77	23
22-24	13	54	36
25-36	35	78	21
37-48	14	57	43
49-60	15	54	46
60+	39	58	42
Unknown	15	73	27
Total	272	69	31

In 63 per cent of these, phrenic paralysis was clearly our first choice and in 9 per cent it was used because there appeared to be a reasonable chance that it would be effective and would make unnecessary a tedious pneumothorax. In 27 per cent, pneumothorax was first attempted and was wholly impossible because of pleural adhesions.

It is interesting that among the 88 arrested cases from the 253 whose preoperative length of sanatorium residence is known to us, 34 had been in a sanatorium for more than six months and 18 for from twelve to more than thirty-six months. The ultimate percentage of arrested cases is 30 for those 102 patients who remained in a sanatorium for less than six months after operation and 34 for those 148 who remained from six to thirty-six months. The exact length of sanatorium residence after operation is unknown to us in 22 patients.

Table I shows the results according to the length of postoperative observation in the cases in which phrenic paralysis was used alone.

TABLE VI

RESULTS WITH REGARD TO LESION

EXTENT OF LESION BEFORE OPERATION	CASES	ARRESTED	IMPROVED	STATIONARY	WORSE	DEAD
		%	%	%	%	%
Unilateral without cavity	50	48	40	8	0	4
Unilateral with cavity	26	42	42	4	4	8
Bilateral without cavity	63	46	38	3	3	9
Bilateral with unilateral cavity	116	23	32	18	9	18
Bilateral cavity, operation on worse side	15	7	27	20	0	47
Unknown	2	0	0	100	0	0
Total	272	34	35	12	5	14

TABLE VII

RESULTS WITH REGARD TO PREOPERATIVE PRESENCE OR ABSENCE OF CAVITY

BEFORE OPERATION	CASES	ARRESTED	IMPROVED	STATIONARY	WORSE	DEAD
		%	%	%	%	%
No cavity	113	47	39	5	1	7
Cavity	159	24	32	17	7	18
Total	272	34	35	12	5	14

The figures suggest that the rest and relaxation of the lung have a continuing favorable influence upon the tuberculosis in that the percentage of good results increases with each year of postoperative observation. We believe that there has not been enough broadening of our indications for the operation during the past few years to account for any but a small part of this phenomenon. Nine of the 272 patients have been observed only for three months after operation, 38 from three to six months, 50 from six to nine months, and 20 from nine to twelve months.

In Tables II to XII, inclusive, are given the data that are of chief interest with respect to phrenic paralysis used independently. The tables are self-explanatory. Only a few incidental remarks will

be made about certain of them. We have made a survey of a large number of articles, exclusive of our own, that have been published during the past fifteen years in which more than a wholly indefinite statement as to results was made. The tuberculosis in 1148, or 60.5 per cent, of the total 1897 cases appears to have been improved or arrested.

The effect of phrenic paralysis upon cavities within the lung is considered from the points of view of the extent of the tuberculosis within the lungs (Table VI), the presence or absence of any cavity (Table VII), the size (Table VIII) and position (Table IX) of the cavity and the effect of the operation upon cough, sputum, fever and weight (Table XI) and hemoptysis (Table XII). We feel that our experience in using Max Pinner's "therapy classification" of cavities is as yet too limited to justify its use in this report.

TABLE VIII

EFFECT ON CAVITY ACCORDING TO ITS SIZE

SIZE OF CAVITY	CAVITIES	CLOSED	SMALLER	SAME	LARGER
	NO.	%	%	%	%
Moth-eaten	11	27	9	45	18
0- 2 cm.	53	65	2	13	20
2- 4 cm.	63	36	27	25	12
4- 6 cm.	32	22	31	12	34
6- 8 cm.	9	12	0	44	44
8-10 cm.	5	0	20	40	40
10 cm. plus	6	0	0	83	17
Total	179*	38	16	24	20

*Although we have been able to determine whether or not 215 cavities closed (see Table IX) we have been able to measure the preoperative diameter of only 179 cavities because some roentgenograms were not available.

TABLE IX

EFFECT ON CAVITY ACCORDING TO ITS POSITION

LOCATION OF CAVITY	CASES	CLOSED	OPEN
		%	%
Upper third	172	40	60
Middle third	36	34	66
Lower third	7	29	71
Total	215*	38	61

The occasional case in which one cavity closed and another remained open is classified as open.

*These include both the cases in which phrenic paralysis was used alone and those in which other measures later became necessary, but the effect of which is not recorded in this table.

Some authors in reporting the proportion of cavities closed by phrenic paralysis, group the cases in which the phrenic operation was used independently and those in which it was added to an unsuccessful pneumothorax. In 38 per cent of our 215 cases with cavity in which the phrenic paralysis alone was given an adequate opportunity, the cavities became closed. In 59 per cent of our 116 cases in which a

TABLE X

INFLUENCE OF LESIONS ON RISE OF DIAPHRAGM AND OF RISE OF DIAPHRAGM ON CLINICAL RESULT

TYPE AND EXTENT OF LESION		CASES	RISE OF DIAPHRAGM AFTER OPERATION									
			0-2 CM.		2-4 CM.		4-6 CM.		6-8 CM.		8-10 CM.	
			RT	LT	RT	LT	RT	LT	RT	LT	RT	LT
			%	%	%	%	%	%	%	%	%	%
Productive	Minimal	21	5	9	28	24	5	19	0	9	0	0
	Moderately Advanced	50	2	2	26	16	14	16	10	6	2	6
	Far Advanced	34	3	0	20	14	20	23	6	9	3	0
	Total	105	3	3	24	17	14	19	6	7	2	3
Exudative	Minimal	9	0	11	33	44	0	0	11	0	0	0
	Moderately Advanced	47	8	2	30	20	17	10	2	2	4	4
	Far Advanced	53	7	5	15	9	20	17	11	11	2	0
	Total	109	7	4	23	16	17	12	7	6	3	2
	Grand Total	214	5	4	24	16	15	15	7	7	2	2
			9%		40%		31%		14%		5%	
Clinical Result			%		%		%		%		%	
Arrested		80	7		38		35		8		10	
Improved		83	6		42		32		16		2	
Stationary		22	18		40		22		18		0	
Worse		5	20		40		40		0		0	
Dead		24	12		41		25		20		0	
Total		214	9		40		31		14		5	

This table is based upon 214 cases. An additional 58 cases cannot be included because either the roentgenograms are inaccessible or the position of the diaphragm is obscure. The probable explanation for the relatively inferior results in the patients whose diaphragms rose from 6 to 10 cm. is that the extensive pulmonary fibrosis that was chiefly responsible for the high diaphragmatic rise, often meant that the lesions were old and the cavities stiff-walled and therefore resistant to closure by mere phrenic paralysis.

phrenic operation was added to an unsuccessful pneumothorax, the cavities became closed. By combining the 331 cases in these two groups, 46 per cent of the cavities became closed and 54 per cent remained open. These figures do not, of course, include any case in which a cavity closed only after a pneumothorax was added to an unsuccessful phrenic paralysis.

Although 68 among 159 cavities that were less than 6 cm. in diameter closed, only one in 20 that were greater than 6 cm. did so. The reason that phrenic paralysis was used in these 20 cases was partly the hope that their cavities might possibly close, but chiefly to delay progression of the tuberculous lesions or to relieve troublesome symptoms in patients in whom pneumothorax could not be produced or in whom other operations were contraindicated at the time.

Measurement of the height of the rise of the diaphragm (Table X) as determined by comparison between the preoperative and last postoperative roentgenograms, was made as follows: The top of a T-square was placed upon the highest points of the transverse processes of the first thoracic vertebrae and the distance measured between this base line and the highest visible point of the hemidiaphragm. We have observed that in those cases in which a phrenic paralysis existed in the presence of a pneumothorax, the diaphragm tended to rise higher after the pneumothorax had been abandoned than in those cases in which phrenic paralysis had been used independently of pneumothorax.

Exhausting and dangerous excessive cough (Table XI) and vomiting are occasionally relieved dramatically through stopping the movement of a diaphragm to which the lung is adherent. Such cases occur most frequently in the presence of a partial pneumothorax.

In Table VII it will be seen that a phrenic operation was performed in 113 patients who had no pulmonary cavity. Among these patients, 23 had no sputum before operation, 50 had negative sputum, 34 had positive sputum and in six the records are incomplete. Table XI shows that 37 patients, with or without cavity, had no sputum before operation. However in only nine cases of moderately advanced and in 21 cases of minimal tuberculosis was the operation performed for strictly protective or prophylactic purposes in patients whose disease was already quiescent or apparently arrested. The results in the nine moderately advanced cases are: Arrested, 66 per cent; improved, 22 per cent; worse, 12 per cent, and in the 29 minimal cases: Arrested, 90 per cent; improved, 5 per cent; worse, 5 per cent (later developed cavity on opposite side).

In Table XII are given the results of phrenic paralysis used for the control of hemoptysis. We prefer this operation to induced pneumothorax as our first choice of collapse procedures for hemoptysis.

TABLE XI

EFFECT ON COUGH, SPUTUM, FEVER, AND WEIGHT

EXTENT OF LESION	CASES	COUGH					SPUTUM					FEVER					WEIGHTS		
		NONE (before oper.)*	DISAPPEARED	LESS	SAME	MORE	NONE (before oper.)*	DISAPPEARED	LESS	SAME	MORE	NONE (before oper.)*	DISAPPEARED	LESS	SAME	MORE	GAIN	SAME	LOSS
		%	%	%	%	%	%	%	%	%	%	%	%	%	%	%	%	%	%
Unilateral without cavity	48	33	34	25	34	6	23	21	42	27	10	48	52	8	24	16	50	21	29
Unilateral with cavity	21	10	58	16	16	10	10	42	37	16	6	61	75	0	12	12	60	5	35
Bilateral without cavity	62	35	50	27	20	2	29	16	40	34	9	53	69	17	14	0	58	13	29
Bilateral with unilateral cavity	96	8	23	37	23	17	5	13	49	16	21	37	55	7	15	23	64	6	30
Bilateral cavity; operation on worse side	14	0	15	28	28	28	7	8	30	23	39	28	30	20	20	30	43	0	57
Total	241	20	38	30	24	14	15	17	43	22	16	45	57	9	16	16	59	10	31

*Figures in "None (before oper.)" columns represent percentages of total 241 cases. Figures in other columns represent the percentages of those patients who had cough, expectoration, or fever before operation.

TABLE XII

EFFECT ON HEMOPTYSIS

EXTENT OF LESION	CASES	HEMOPTYSIS				CASES	STREAKING			
		CEASED %	IMPROVED %	SAME %	WORSE %		CEASED %	IMPROVED %	SAME %	WORSE %
Unilateral without cavity	6	83	0	0	17	2	50	0	50	0
Unilateral with cavity	1	0	0	0	100	1	0	100	0	0
Bilateral without cavity	2	50	0	50	0	6	83	0	17	0
Bilateral with unilateral cavity	8	75	12	12	0	4	50	0	25	25
Bilateral cavitation; oper. on worse side	2	33	0	0	67	0	0	0	0	0
Total	20	65	5	10	20	13	61	7	23	7

TABLE XIII

COMPLEMENTARY COLLAPSE PROCEDURES AND FINAL RESULTS

EXTENT OF LESION	CASES	PROCEDURES*							RESULTS					
		PNEUMOTHORAX	THORACOPLASTY	PARAFFIN EXTRAPLEURAL PNEUMONOLYSIS	PECTORAL M. EXTRAPERIOSTEAL PNEUMONOLYSIS	BAG EXTRAPERIOSTEAL PNEUMONOLYSIS	INTERCOSTAL NEURECTOMY	SCALENECTOMY	ARRESTED	IMPROVED	STATIONARY	WORSE	DEAD	NOT TRACED
		%	%	%	%	%	%	%	%	%	%	%	%	%
Unilateral without cavity	2	50	50	0	0	0	0	0	0	50	0	0	0	50
Unilateral with cavity	18	55	50	6	0	6	6	0	28	44	0	0	17	11
Bilateral without cavity	10	50	10	20	0	0	10	20	0	60	10	20	0	10
Bilateral with unilateral cavity	29	60	27	28	3	0	3	0	7	55	4	10	17	7
Bilateral cavity; operation on worse side	3	33	0	33	0	0	0	33	0	100	0	0	0	0
Basal cavitation	4	100	0	50	0	0	0	0	0	75	0	0	0	25
Total	66	57	30	20	2	2	5	3	11†	56	3	7	12	11

*The total number of these operations is 77. Some of the patients especially those having had an unsuccessful pneumothorax, had more than one collapse measure, other than phrenic operation and, therefore, the horizontal rows of figures may total more than one hundred per cent.

†Few because too soon since operation. Many in *Improved* group aresputum negative and probably will be *Arrested* eventually.

Three cases of frank hemoptysis and two cases of streaking occurred during the course of pneumothorax, and phrenic paralysis was used to control the bleeding. It was completely successful in all but one case of frank hemoptysis.

One patient had his first streaking, but no hemoptysis, within a month after a Goetze phrenic operation. One additional patient had postoperative streaking and another an hemoptysis, but both were operated on too recently to be included in this study. None of these patients died.

There were 14 patients who had had no hemoptysis before the phrenic operation but did have for the first time some months after operation. In three it was severe; death resulted in two and in one induced pneumothorax was successful in controlling the bleeding. There is no evidence that the phrenic operation was responsible for the hemoptyses in these 14 patients, except through failure to heal the tuberculosis promptly.

The phrenic operation was not the cause of death in any case. We will report here the causes of death in the 38 cases among the 272 in which phrenic paralysis was used independently. In 34 cases death was due to gradual progression of tuberculosis in the lungs, and often in other organs. In the great majority of these cases the disease was far advanced at the time of operation. In many of them there was no reasonable hope of arrest of the disease from phrenic paralysis but only of palliation. One patient with an exudative actively progressing lesion died one week after operation, one died one and a half months after operation, four died from two to two and a half months after operation and the others, except one whose time of death is unknown to us, died from three and a half to thirty-two months after operation. The 33 patients whose death was due to progressive tuberculosis include three with a terminal severe hemoptysis at eight, nine and thirteen and a half months after operation and two with terminal meningitis at two and a half and nine months and one at four months from thrombophlebitis and spreading of tuberculosis throughout both lungs with spontaneous pneumothorax, following pregnancy. The four deaths not due to progressive tuberculosis include two from uremia eight and thirteen months after operation; in one patient there had been improvement in the pulmonary lesions and in the other no apparent change. The third patient died from "cardiac failure" two and a half months after operation and the fourth patient from "pneumonia" thirty months after operation, after having been discharged from a sanatorium as an apparently arrested case.

Twenty-six patients in whom phrenic paralysis had been used independently could not be followed after their discharge from the hospital or sanatorium a few days, weeks or months after operation. The

last reports of these patients show that 4 per cent (one patient) were arrested, 46 per cent were improved, 50 per cent were unimproved, and none was worse.

UNSATISFACTORY PNEUMOTHORAX FOLLOWED BY PHRENIC OPERATION

We have traced 181 patients who had had induced pneumothorax as the first collapse therapy procedure and in whom a phrenic paralysis was subsequently added. These patients had had a trial of pneumothorax alone for at least two months.

In 65 of these cases, the phrenic operation was performed for noncavernous lesions either to increase an unsatisfactory collapse or as a protective measure to give the patient the continuing benefits of rest and relaxation of the diseased lung at the time of abandoning a satisfactory pneumothorax or upon discharge of the patient from a sanatorium. The results in these 65 patients are: 42 per cent arrested, 50 per cent improved (many of the improved patients are fully expected to enter the arrested group when the requisite number of months of the present absence of symptoms have elapsed), none stationary or worse and 7 per cent dead.

Phrenic operation was added to an unsatisfactory pneumothorax in 116 of the 181 patients because of the continuing presence of cavernous lesions. The addition of the phrenic paralysis resulted in complete closure of the cavity in 59 per cent (69 patients) of these patients (the cavity having closed in less than two months in 19 per cent) and the cavity remained open in 40 per cent (47 patients). The results in the 69 patients with closed cavities are 26 per cent arrested, 63 per cent improved, none stationary, 6 per cent worse and 4 per cent dead. Two of the 44 patients listed as improved had had their pulmonary cavities closed by the combination of pneumothorax and phrenic paralysis but a pneumothorax-empyema required a later thoracoplasty which solidly closed both empyema cavities. Two other patients who are classified as improved had had gomenol substituted for the pneumothorax and three had bilateral pneumothorax.

Fifty-one per cent of the 47 patients whose cavities remained open after phrenic operation was added to a pneumothorax have persistently open cavities or died before the cavities closed. Forty-nine per cent of the 47 patients have had further surgery used in an attempt to close the cavities. In 26 per cent (13 per cent of the 47 patients in whom the cavity remained open after phrenic operation) this was unsuccessful and the cavities are still open, whereas in 74 per cent (36 per cent of the whole group) the cavities were closed by additional surgery. The ultimate clinical results in the 23 cases who had further surgery are: 17 per cent arrested, 56 per cent improved, 9 per cent stationary, none worse, and 18 per cent dead. The further surgical measures used were: Closed intrapleural pneumonolysis in six pa-

tients, open pneumonolysis in one, extrapleural pneumonolysis with paraffin filling in one, and with rubber bag filling in one, thoracoplasty in 14.

In addition to the 181 patients in whom pneumothorax was followed by phrenic operation, there were seven who could not be traced after their discharge from hospital or sanatorium. Fifty-seven per cent of their cavities were closed at the time of discharge; the tuberculosis in 14 per cent was arrested, in 43 per cent improved and in 43 per cent unimproved.

UNSATISFACTORY PHRENIC FOLLOWED BY OTHER COLLAPSE MEASURES

In 66 patients phrenic paralysis failed to produce satisfactory clinical results and therefore some other collapse therapy measure or measures were later used as supplementary procedures.

The interval between the phrenic operation and the supplementary procedure was from one to two months in 15 per cent of the 66 cases, two to four months in 25 per cent, four to six months in 16 per cent, six to eight months in 14 per cent, eight to ten months in 6 per cent, ten to twelve months in 4 per cent, twelve to eighteen months in 11 per cent and more than eighteen months in 10 per cent.

A few of the reasons for having done a phrenic operation in advance of the major procedure are as follows: In five cases it was hoped that a thoracoplasty might be averted, but the hope was not fulfilled in these particular cases, though it was in others which are entered under the successful results from phrenic paralysis used independently. In several cases it was used to prepare patients who were in poor general condition for a thoracoplasty or in whom active lesions in the contralateral lung were considered too active at the time of the phrenic operation to justify a thoracoplasty then. In several cases the phrenic paralysis was used to aid in the collapse of large cavities and extensive lesions. In one patient with a temporary phrenic interruption, return of movement of the diaphragm in a distant city was not followed by a repetition of the phrenic nerve interruption and the small cavity steadily enlarged so that a major operation became necessary. Only two of seven basal cavities were closed by phrenic paralysis; of the remaining five cases, four were suitable for some supplementary collapse therapy measure.

An unsuccessful attempt was made to induce pneumothorax before the phrenic operation was performed in 26 per cent of the 66 patients and no such attempt was made in 74 per cent. In the latter group of 49 patients pneumothorax was attempted after the phrenic operation in 38. In these patients it was technically satisfactory in 61 per cent and unsatisfactory in 39 per cent.

The different operations used in conjunction with phrenic operation in the 66 patients are given in Table XIII with reference both to the

different distributions of the tuberculous lesions and to the final results. Seventy-seven operations, other than phrenic nerve interruption, were used for the 66 patients because a few of the patients required more than one operation, usually on account of a pneumothorax's proving to be unsatisfactory.

PHRENIC AND OTHER COLLAPSE MEASURES USED CONCOMITANTLY

Mention was made in the seventh paragraph of this article of 67 cases in which phrenic paralysis was so soon followed by (occasionally preceded by and, in the case of scaleniectomy, accompanied by) thoracoplasty, extrapleural pneumonolysis, multiple intercostal neurectomy or scaleniectomy that they were intended to complement one another. The phrenic operation had been performed without the intention of waiting for an independent favorable effect of the phrenic operation because the lesion was usually so extensive that it was obvious that more than the phrenic paralysis would be needed. It is apparent that the results in these cases, except perhaps those having scaleniectomy, owe far more to the major operation used than to the phrenic operation and as the results should be and are approximately representative of each major operation, they will not be listed here.

SUMMARY

1. Of the 654 patients who have had a phrenic operation for phthisis from three months to six years before this report was prepared, the present status of 612 or 93.5 per cent is known.

2. Data are presented with regard to the types of operation used, operative and postoperative complications, effect upon tubercle bacilli, sputum, hemoptysis, cough, fever and weight. Results, according to the criteria of the National Tuberculosis Association, and with respect to many pertinent factors, are reported separately for groups of cases in which phrenic operation has been used independently and in various combinations with supplementary and with complementary operations. For the group in which phrenic operation was used independently, results are presented with reference to the length of postoperative observation, height of the rise of the diaphragm, type and extent of the lesions, severity of symptoms, ability to work, age of the patients and of their lesions, the presence or absence of a pulmonary cavity and the size and position of the cavity.

3. Phrenic operation was used alone in 302 patients and, in addition, in three cases of the childhood type of tuberculosis and in three cases on both sides, but on the second side only after diaphragmatic motion returned following a temporary interruption of the nerve on the first side. Two hundred seventy-two of the 302 patients have been traced; 34 per cent are apparently cured or arrested, 35 per cent are apparently arrested, quiescent or improved, 12 per cent are sta-

tionary, 5 per cent are worse and 14 per cent are dead. More than half of the cured or arrested patients had been under treatment in sanatoria for from eight to thirty-six months before the operation had been performed. We have collected from many articles 1897 cases of phrenic paralysis in which 60.5 per cent have been reported as cured or improved by the operation.

4. There were 215 cases with pulmonary cavity in which phrenic paralysis alone was given an adequate opportunity to close the cavity. It succeeded in 38 per cent. Fifty-nine per cent of the 116 cavities that had remained open after a two or more months' trial of pneumothorax, were closed by a subsequent phrenic paralysis. By combining these two groups, 46 per cent of 331 cavities were closed by phrenic paralysis. These figures do not include any case in which a pneumothorax closed a cavity after an initial phrenic operation had failed to do so.

5. Sixty-five of the 181 traced patients, in whom a phrenic paralysis was used following an unsatisfactory pneumothorax had had no cavity; 42 per cent are arrested, 50 per cent are improved and 7 per cent are dead. The cavities of 59 per cent of the 116 patients who had had cavities are closed. Fifty-one per cent of the 47 cavities that remained open are still open or the patients are dead and 49 per cent have been closed by thoracoplasty, extrapleural pneumonolysis, multiple intercostal neurectomy or scaleniectomy.

6. An initial phrenic paralysis failed to cause satisfactory improvement in 66 patients, for whom pneumothorax, thoracoplasty or other supplementary operation was subsequently added. The results in these cases are: 11 per cent arrested, 56 per cent improved (many of these are without any symptoms of tuberculous activity and will automatically enter the arrested group when they have been symptom-free for the requisite time), 3 per cent stationary, 7 per cent worse, 12 per cent dead and 11 per cent not traced. Pneumothorax was first attempted but was impossible in 26 per cent of the 66 patients; it was finally undertaken in 38 of the 49 patients in whom it had not first been tried and in 61 per cent of these it was technically satisfactory. These figures seem to indicate that phrenic paralysis does not in itself cause pleural adhesions that might prevent the subsequent induction of a satisfactory pneumothorax.

7. In 67 patients phrenic operation was used concomitantly with thoracoplasty, extrapleural pneumonolysis, intercostal neurectomy or scaleniectomy. The clinical results obviously owe more to the major operations (except perhaps scaleniectomy) than to the phrenic operation.

8. In the entire series of 654 patients, phrenic paralysis fulfilled our expectation of it, which ranged from mere palliative symptomatic

relief to complete arrest of the tuberculosis, in 58 per cent, partly fulfilled it in 6 per cent, failed to fulfill it in 30 per cent, and in 5 per cent the result is unknown to us.

9. In the entire series, phrenic paralysis was neither directly nor indirectly responsible for the death of any patient. The only severe, permanent complication of the operation was a dyspnea that has persisted for eighteen months. In one patient, the operation was immediately followed by extension of a few active lesions in the contralateral lung, which was then checked by induced pneumothorax.

10. Our best results have been in limited lesions that were relatively fresh and that contained fibrous tissue that was young enough to be still capable of considerable contraction. Our least satisfactory results have been in extensive fibrotic lesions with large stiff-walled cavities.

11. As phrenic paralysis has been the principal factor responsible for arrest of the tuberculosis in 34 per cent and for improvement in 35 per cent in our patients for whom the operation was used independently and as the only severe permanent complication in 654 patients was marked dyspnea in one patient, we are convinced that phrenic paralysis is of genuine value. The treatment of phthisis would suffer greatly if the operation were not available.

THE EFFECT OF PHRENICECTOMY UPON COUGH AND EXPECTORATION

A Study Based Upon the Elimination of Lipiodol and Foreign Bodies From the Lungs*†

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OPINIONS still differ regarding the effect of a paralyzed diaphragm upon cough and expectoration. Among many there is prevalent the idea that both are facilitated by the various operations which are now being performed frequently upon the phrenic nerve. To quote only a few such positive statements which are to be found in the literature, "The undoubted improvement which frequently follows the operation (phrenicectomy for bronchiectasis) must be attributed to the greater ease of expectoration which prevents the stagnation and decomposition of the secretions in the dilated tubes." (Wall.) "The striking facilitation of cough after paralysis of the diaphragm is interesting. It is due to the greater compression exerted upon the lungs by the contraction of the abdominal muscles with the diaphragm flaccid. Normally this pressure is limited by the tone of the diaphragm." (Straub.)

Sauerbruch, Matson, and others have expressed similar opinions. Yet, as a matter of fact, from a purely clinical standpoint there is often evidence that cough and expectoration are made more difficult, particularly in the presence of bronchial dilatations which concern us most. Referring to some of the clinical experiences of others we note that Cooper found lessening of cough in over 40 per cent of cases. Bronfin and Chernyk have tabulated the results of treatment by phrenicectomy in 183 patients nearly all of whom had pulmonary tuberculosis. They noted a palliative effect, decrease in cough and sputum, and greater ease of expectoration in from 40 to 50 per cent of their cases. Their tabulated results suggest that phrenicectomy may exert either a favorable or an unfavorable effect upon cough and expectoration, regardless of whether the lesion is located in the base or the apex. Moore states that phrenicectomy usually makes cough and expectoration easier but adds that the reverse may also be true.

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Wilson has reported his experiences in 18 cases of bronchiectasis and 21 cases of pulmonary tuberculosis treated by phrenic evulsion. He found that expectoration was made easier in 4, more difficult in 3, and was unchanged in 11 in the bronchiectasis series. Similarly in the 21 tuberculous patients cough was made easier in 5, more difficult in 3, was unchanged in 12, and one had no sputum.

Berry is among those who conclude that phrenicectomy does not always facilitate cough and expectoration; occasionally it has the opposite effect. He was able to collect from the literature and from his own series, cases in which "the operation so hindered cough and drainage, with resultant decrease in sputum, that the secretions puddled in the lung and an extensive fatal pneumonia developed."

This difference in opinion and the lack of experimental evidence for either side prompted us to investigate the effect of a paralyzed diaphragm upon the expulsive function of the lungs. To do this we concerned ourselves chiefly with the effects of injection and the rate of disappearance of lipiodol before and after phrenicectomy.

Our method of investigation makes it imperative that we consider how lipiodol is eliminated from the lungs under normal conditions. Before so doing, however, we should like to define briefly just what is meant by cough and by expectoration and also to indicate the rôle which the normal diaphragm is said to assume in the execution of each.

The Mechanism of Cough.—Cough may be either reflex or voluntary and is essentially a forced expiration. "The abdominal muscles contract, bringing about an increase in intraabdominal pressure which is transmitted through the semirigid diaphragm into the thoracic cage. The intercostal muscles, aided by the abdominal and lumbar groups, tend to elongate the bony framework of the thorax and thereby markedly decrease its average diameter. The accompanying contraction and fixation of the diaphragm further narrow the chest cavity and complete the contraction of the boundaries of the thoracic cage, except for the upper segment represented by the neck. This is held rigid by the long muscles of the neck, leaving only the exit through the trachea unaccounted for. The sum total of the contraction of all the muscles involved results in a sudden diminution of intrathoracic capacity, with a corresponding concentric rise in intrathoracic pressure." (Archibald and Brown.) In addition to the above, mention must also be made of the fact that Wenckebach stresses the importance of the lateral bundles of the latissimus dorsi muscles which are said to compress the thorax during cough.

The glottis too plays a special rôle, for "When a sufficiently high intrapulmonic pressure is attained, the glottis opens and the sudden change in pressure causes a forcible blast of air." (Macleod.) The velocity of this blast of air as it passes through the various segments of the human respiratory tract has been calculated by Rohrer. He

found that it varied from 0.5 to 2 meters per second in the respiratory bronchiole, and from 50 to 120 meters per second at the glottis. The velocities in the upper air passages during cough were compared with wind velocities and it was found that they were comparable to the wind velocities of severe storms. Rohrer has further pointed out that the coughing process is suitable for the expectoration of secretions from bronchi over one millimeter in diameter and of cavities communicating with the bronchial system. Cough is apparently not adapted to the removal of secretions from the smaller divisions of the bronchial tree.

The Mechanism of Expectoration.—Expectoration which implies the expulsion of material from the mouth and air passages is according to Wall "effected by raising the intrapulmonary pressure and then suddenly allowing the air to escape and carry with it any peccant matter that may be lying in the air passages." This definition makes expectoration dependent upon cough or some cough-like forced expiration. As Blackader has pointed out, however, such a definition is incomplete, as it does not contain any reference to "the movement of the ciliated cells in the air passages which effect the first act of expectoration." It is our opinion, based upon the work of Bullowa and Gottlieb, Heinbecker, Reinberg, Macklin, and others, that at least one more factor must be of importance, namely, movements of the bronchi. It is hardly necessary to mention that gravity is concerned in expectoration and is made use of in postural drainage; further, that material raised from the tracheobronchial tree to the pharynx may be either expectorated or swallowed.

Rôle Played by the Diaphragm in Cough and Expectoration.—That the diaphragm is not absolutely essential to cough and expectoration can be readily demonstrated by observing fluoroscopically the act of coughing when one-half of the diaphragm is paralyzed.

Our point, however, is whether cough and expectoration are enhanced or handicapped by paralyzing the diaphragm. Those who contend that phrenicectomy facilitates cough offer as their reason the loss of diaphragmatic tone. But, on the other hand, as each cough is usually preceded or followed by a deep inspiration, and as the diaphragm is an important muscle of inspiration, it is probable that in a succession of coughs the excursion of the diaphragm will in some measure condition the mechanical efficiency of cough.

One may rightly ask if it matters whether the diaphragm is in a position of inspiration or expiration (position of the paralyzed diaphragm) when the act of coughing is initiated. In this connection the recent studies on bronchial obstruction and collateral respiration by Van Allen and his collaborators are interesting and instructive. In their investigation, the importance of the column of air behind the

obstruction has been emphasized. "The available air is the ram that drives through the bronchi and trachea in coughing." It is clear that the available air is fundamentally related to the depth of inspiration, and although inspiration is made up of both intercostal and diaphragmatic components, the importance of the diaphragm in inspiration cannot be disregarded. In this sense it may be said that interference with cough and expectoration subsequent to phrenicectomy appears to be theoretically possible.

The Elimination of Iodized Oil From the Lungs.—Iodized oil may be removed from the lungs by several mechanisms some of which are included under the terms cough and expectoration. The current of air passing through the air passages in cough or forced expiration is undoubtedly of importance in expelling oil which is situated in the trachea or bronchi. On the other hand, cough may force some of the oil deeper into the lung, producing an alveolar distribution. The movements of the bronchi, consisting of the changes in length and width which take place in each respiratory cycle, and also peristalsis or peristaltoid movement, appear also to be capable of expelling oil. The significance of ciliary movement in the removal of oil has received little attention but we have demonstrated by placing oil upon the mucosa of the excised trachea of dogs that it may be transported at the rate of 1 cm. in 80 seconds when the trachea is in a horizontal position. These methods suffice for removal of oil in the tracheobronchial tree but the oil which reaches the alveoli must be removed to some extent by other means.

Phagocytosis of iodized oil occurs in the lungs. In experiments of Fried "small amounts of oils injected into animals' lungs by way of the trachea cause a reaction confined essentially to the cells lining the walls of the alveoli. These cells 'swell,' acquire a foamy cytoplasm, proliferate instantaneously, phagocytose the oil and finally detach themselves from the pulmonary septums to form the 'inflammatory' intraalveolar exudate. They also form giant cells of the Langhans type." Some of the "lipoidophage" cells may reach the bronchi and be expectorated. Finally, the iodized oil may be acted upon by lipolytic ferments, iodine absorbed into the blood stream and excreted in the urine as experiments by Sicard, Fahre and Forestier and by Ballon and Curtis have demonstrated.

The rate of elimination of oil from the bronchi has been investigated by Jacobaeus, Selander and Westermark who attempted to develop a clinical functional test of the emptying capacity of the bronchi. Their method consisted of introducing lipiodol stained with Sudan III and observing the expectoration of the oil. The first phase in which a layer of the red-stained oil formed in the sputum glass lasted in normal subjects four to eight hours. Then there sometimes followed a

transition-phase lasting eight to twelve hours in which lumps of oil were coughed up. This phase was also lacking. The second phase often lasting several days was characterized by streaks or spots of lipiodol in the sputum. The first phase was thought to be an expression of the emptying capacity of the bronchi, the second phase corresponded to the emptying of the finest bronchioles and alveoli, a process much slower and more incomplete. These observations on expectoration time are of interest in the interpretation of the results of our experiments.

Experiments.—The experiments were concerned chiefly with the elimination of lipiodol from the lung in the presence of a paralyzed diaphragm as compared with its elimination in the presence of a normal diaphragm. The rate of disappearance of the oil was determined by means of fluoroscopic examinations and serial roentgen ray films. The effect of phrenicectomy upon the ability to cough up foreign bodies was also studied.

Technic of Phrenicectomy.—A transpleural phrenicectomy was performed upon 8 dogs. The thoracic approach was adopted at first in order to make sure that the entire phrenic nerve would be interrupted. The possibility that additional factors had been introduced by opening the pleura was, however, excluded by performing the phrenicectomy in the neck on 3 dogs.

The thoracic operation was performed transpleurally through a short intercostal incision under ether anesthesia administered with positive pressure. The nerve was picked up as it coursed over the pericardium and a segment 2 or 3 cm. long was excised. Interrupted sutures were placed about the ribs and the lungs were inflated as these sutures were tied. The muscles and the skin were then approximated with interrupted sutures.

Phrenicectomy in the dog through a cervical incision, although technically more difficult, was satisfactory. An oblique incision was made along the posterior border of the sternomastoid muscle, the brachial plexus was exposed, and the phrenic nerve coursing downward was identified. The nerve was then retracted gently and all communicating branches were divided as the nerve was followed downward. When all of the communicating twigs had been divided, the phrenic nerve was cut across. In one instance it was evulsed.

Technic of Oil Injections.—The lipiodol injections were usually made as follows: The dog first received one-half to one and a half grains of morphine sulphate subcutaneously. After an interval of about one hour, under laryngoscopic and fluoroscopic control a brass tube was inserted into either the right or left main bronchus and a measured amount of the iodized oil injected. In the experiments in which both sides were injected the tube was then withdrawn slightly and inserted into the opposite main bronchus where an equal amount of oil was injected. A roentgen ray film was then taken. The disappearance of the oil from the lungs was followed fluoroscopically and by means of serial roentgen ray films. The observations were continued until all of the oil had disappeared from one side. In taking the films posteroanterior exposures were made at the end of inspiration with the dog lying on its back.

This general plan was departed from in carrying out some special observations. In five experiments (Series IV) the oil was merely introduced into the trachea and permitted to flow by gravity into the right or left bronchial tree by tilting the animal to one side or the other.

The 24 experiments upon 11 dogs may be grouped as follows:

Series I. (Eight experiments.) A preliminary unilateral phrenicectomy was performed. After permitting adequate time (two to three weeks) for complete recovery from the operation, oil was injected into both the right and left lower lungs. The rate of disappearance of the oil from the paralyzed side was compared with that of the normal side.

Series II. (Two experiments.) A preliminary oil injection was made on one side and the rate of elimination of the oil was determined. A phrenicectomy was then performed upon the same side and the oil injection repeated. The rates of disappearance of the oil before and after phrenicectomy were compared.

Series III. (One experiment.) The effect of bilateral phrenicectomy was investigated. The dog was subjected to a unilateral phrenicectomy first (Series I) and the disappearance of oil from the normal and paralyzed sides was compared. Subsequently the other half of the diaphragm was paralyzed and the experiment repeated.

Series IV. (Thirteen experiments.) In this series of experiments attempts were made to evaluate the immediate effects of cough after introducing lipiodol or solid foreign bodies into the bronchi. The animals were mechanically stimulated to cough in some experiments but in others the effect of the foreign material placed in the bronchial tree sufficed to induce coughing. In 3 instances lipiodol was placed in the bronchi on the side of the normal diaphragm, and in two instances on the side of the paralyzed diaphragm. In 2 experiments lipiodol was introduced on both sides and in 6 experiments foreign bodies consisting of shot or barium capsules were introduced into the bronchi through a bronchoscope. The ability of the animals to expel material from the bronchi by coughing was studied.

RESULTS OF THE EXPERIMENTS

Series I. The results of the experiments in this series in which unilateral phrenicectomy was followed by bilateral injection of the oil are tabulated (Table I). Retardation of the elimination of oil was found to occur on the side of the paralyzed diaphragm in 7 of the 8 experiments. In only one instance were the results questionable and in no instance did the oil disappear first on the side of the phrenicectomy. The results were just as striking in those animals in which the phrenicectomy was performed in the neck as in those in which it was performed in the chest. Figs. 1 and 2.

Fluoroscopic observations revealed in some of these experiments that when equal amounts of oil were injected on the two sides much of the oil was coughed up almost immediately on the side of the active diaphragm. On the side of the paralyzed diaphragm the oil was not coughed up so effectively and more of it entered the alveoli to produce

the roentgen ray appearance of drowned lung. Cough in these animals occurred as a result of the presence of the brass injecting tube in the trachea or bronchus.

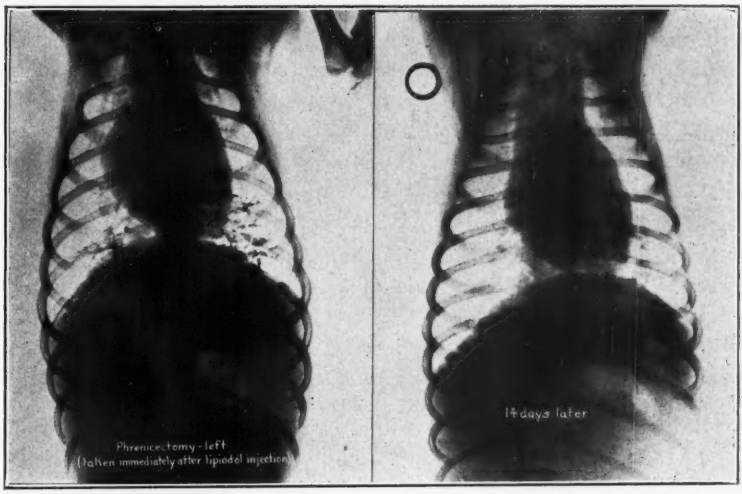

Fig. 1.—Left phrenicectomy was performed in the neck. Elimination of lipiodol was retarded on the side of phrenicectomy.

Fig. 2.—Right phrenicectomy was performed in the neck. Elimination of lipiodol was retarded on the side of phrenicectomy.

In other experiments no marked immediate difference between the two sides was noted but it was observed that the oil disappeared more slowly on the side of the paralyzed diaphragm.

Series II. In the 2 experiments in which the rate of elimination of oil was determined on one side before and after phrenicectomy it was found that the elimination was retarded definitely in one instance and slightly in the other subsequent to phrenicectomy.

Series III. Bilateral phrenicectomy resulted in delayed elimination of the oil. In this animal a previous study made after unilateral phrenicectomy showed that the oil disappeared from the side of the active diaphragm in fourteen days, from the paralyzed side in fifty-five days. Following the bilateral phrenicectomy a considerable quantity of the oil remained on both sides thirty days after the injection. This dog was mangy and died before any further studies could be made.

Series IV. The results obtained in experiments which were undertaken to determine the immediate effect of cough upon the elimination

TABLE I

EFFECT OF PHRENICECTOMY UPON THE ELIMINATION OF LIPIODOL FROM THE LUNG

(Preliminary Phrenicectomy Followed by Injection of Oil)

NO.	AMOUNT OF LIPIODOL INJECTED INTO EACH LUNG	SIDE OF PHRENICECTOMY	LUNG FROM WHICH OIL DISAPPEARED COMPLETELY FIRST	NO. OF DAYS	RETARDATION OF ELIMINATION, RESULT OF PHRENICECTOMY
B-50	3 c.c.	Left	Right	49	Marked
B-20	5 c.c.	Right	Left	63	Definite
A-12	2½ c.c.	Left	Right	88	Slight
B-46	3 c.c.	Right			Not appreciable (63 days)
B-48	6 c.c.	Right	Left	36	Marked
524	3 c.c.	Left	Right	36	Marked
344	4 c.c.	Right	Left	14	Definite
B-6	3 c.c.	Left	Right	14	Definite

of material from the bronchial trees of dogs with unilateral phrenicectomy corroborated the fluoroscopic findings noted in Series I; some exceptions were observed. In one experiment in which a bilateral filling of the bronchi was attempted by introducing lipiodol into the trachea and changing the posture of the animal to permit the oil to flow first into one side and then into the other the bronchi failed to fill properly on the side of the active diaphragm. In a second experiment of the same type a uniform distribution of the oil was obtained. When this animal was stimulated to cough, there resulted an alveolar distribution of the oil. No striking difference between the two sides was noted. This experiment merely demonstrates again that excessive coughing may force the oil deeper into the lungs.

In 3 dogs with unilateral paralysis of the diaphragm the oil was injected into the bronchi only on the side of the normal diaphragm. In each of these three experiments after the animals had been stimu-

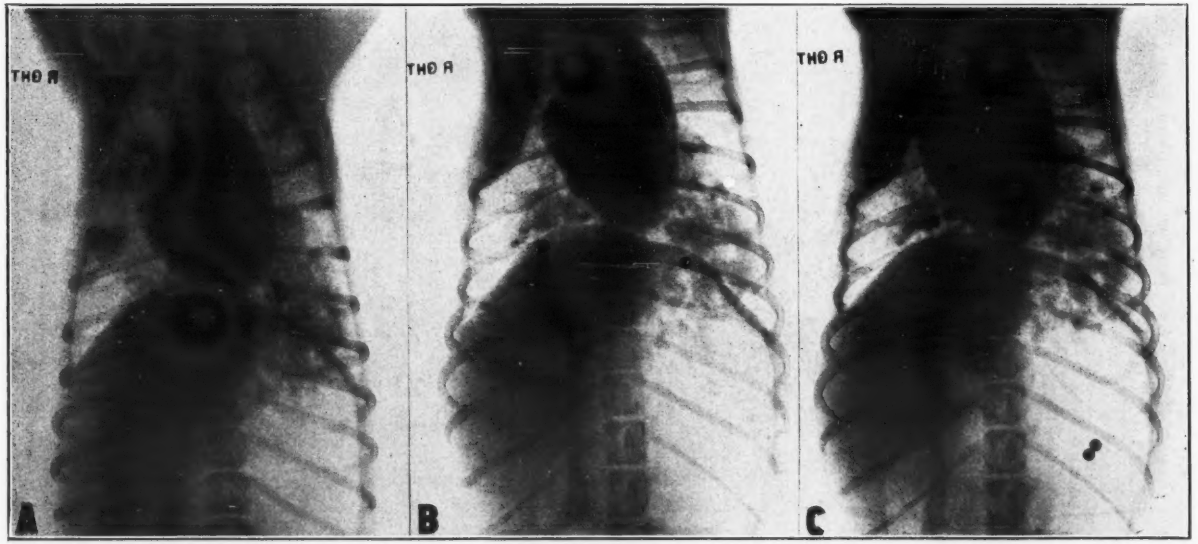

Fig. 3.—Two foreign bodies (lead shot) were inserted on each side through a bronchoscope. (Phrenicectomy, right.) Film A was taken immediately. Film B was taken after dog had been stimulated to cough for fifteen minutes. One of the shot had been coughed over to the side of the paralyzed diaphragm. A few minutes later there were again two shot on each side. Film C taken forty minutes after introducing the foreign bodies reveals that two of them remained on the side of the paralyzed diaphragm and two have been coughed out of the lung on the side of the normal diaphragm. They are seen in the stomach. Some oil is present in the lung from a previous experiment.

lated to cough some of the oil was found in the opposite lung. In two experiments in which the oil was introduced into the lung on the side of the paralyzed diaphragm the results were inconstant. In one, cough produced by mechanical stimulation failed to transport oil to the opposite lung but in the other a small amount of oil gained access to the lung on the side of the normal diaphragm. In this last experiment oil was observed in the trachea when the first film was taken and for that reason it cannot be assumed that it was coughed from one side to the other. The dogs were kept lying on their backs to prevent any gravitational flow of the oil. The results suggest that oil is more readily coughed into the paralyzed side, but as the oil sometimes entered the opposite lung before actual coughing occurred, it is apparent that the act of coughing is not absolutely essential to the transport of oil from one side to the other.

Foreign bodies (shot or barium capsules) introduced bronchoscopically in 6 dogs were eventually coughed up whether placed in the bronchi on the side of the normal or on the side of the paralyzed diaphragm, but they were more efficiently expelled from the side of the active diaphragm. In the first experiment of this type in which two barium capsules were placed in each lower bronchus eventually all four were found in the bronchi on the side of the paralyzed diaphragm. Two subsequent experiments using lead shot gave no conclusive results. As the posture of these animals had not been controlled three experiments with lead shot were repeated, the dogs being kept lying on their backs throughout the periods of observation. In each of these three experiments in which foreign bodies were placed in the bronchi of the two sides, those on the side of the active diaphragm were coughed up more efficiently. In each of the three at some period in the experiment a foreign body was coughed from the side of the active diaphragm to the side of the paralyzed diaphragm. Coughing of a foreign body from the side of the paralyzed diaphragm to the side of the active diaphragm was observed in only one of the experiments (Fig. 3) and in this instance the evidence was clearly in favor of greater efficacy of the cough on the side of the normal diaphragm.

COMMENT

In our experimental studies no support was found for the widely accepted belief that paralysis of a leaf of the diaphragm facilitates cough and expectoration. On the contrary the experiments reveal that the elimination of iodized oil from the lungs is retarded; also that the coughing up of foreign bodies is handicapped by phrenicectomy.

Of the several mechanisms responsible for the removal of oil from the lungs it is difficult to state with certainty which is primarily concerned in this retardation. However, it is unlikely that ciliary movement, phagocytosis, or the action of lipolytic ferments are influenced

by paralyzing the diaphragm. The experiments dealing with the immediate effect of coughing upon the elimination of material from the bronchial tree provide further evidence which suggests that on some occasions coughing may actually drive material, such as lipiodol, more deeply into the lung. That phrenicectomy handicaps cough is also suggested. The limitation of movements of the bronchi resulting from the elevation and immobility of the paralyzed diaphragm also appears to be of importance in the delayed emptying time of the lung noted in our experiments.

In this connection the phenomenon of paradox motion and the aspiration of "bronchial contents" deeper into the lung must be given consideration. According to Brauer when a lung is compressed and is at rest, as in pneumothorax, aspiration into the lung is diminished, but in the presence of paradox movement the tendency to aspiration into the lung is increased. Lemon has also referred to the aspiration of material into the lung in the presence of a unilateral paralysis of the diaphragm. He found that aspiration of particulate matter of any type occurs just as readily into the side of the paralyzed diaphragm as into the side of the normal diaphragm.

Whether the differences noted on the two sides in our lipiodol experiments are due entirely to less effective removal of the oil or in part to actual aspiration into the lung after phrenicectomy cannot be stated with certainty.

The impairment of cough by phrenic paralysis can be best explained on the basis of a diminution in the amount of available air resulting from the partial immobilization and deflation of the lung.

CLINICAL APPLICATION

Caution must be exercised in applying to clinical problems our experimental results upon normal animals.

We are unable, however, to confirm the teaching that artificial paralysis of the diaphragm provides a means of increasing the mechanical efficacy of cough and expectoration. It is to be noted further that the alleged facilitation of cough and expectoration following phrenicectomy in many patients is not to be interpreted necessarily in mechanical terms but may be due to the influence of rest and relaxation of the lung upon pulmonary disease.

We found a delay in the elimination of oil from the lungs and decreased efficiency of the cough mechanism following phrenicectomy. Of equal significance, however, is the observation that even in the presence of a paralyzed diaphragm the cough mechanism can be adequate enough to expel foreign bodies from the bronchi. One can only suggest therefore that the progression of disease sometimes noted after phrenicectomy, particularly in cases of bronchiectasis and pulmonary suppuration, is due to an impairment of cough and expectoration.

CONCLUSIONS

1. The elimination of lipiodol from the lungs is retarded by phrenicectomy.

2. The ability of dogs to cough foreign bodies from the bronchi is impaired but not lost as a result of phrenicectomy.

3. No evidence was found to support the contention that cough and expectoration are facilitated by paralyzing the diaphragm.

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DISCUSSION OF PAPERS BY DRS. DECKER; NEHIL AND ALEXANDER; AND CARLSON, BALLON, WILSON AND GRAHAM

DR. HOWARD LILIENTHAL (NEW YORK CITY).—Let me confine my remarks to very few of the aspects of these three extraordinary and most interesting papers in the order in which I made notes.

I am honored that Dr. Decker has adopted the method of operating which I described and which I have followed for a number of years.

Two points about this operation should be noted. One is the matter of the immediate dressing.

I apply a thick pad over the subclavian triangle region, and strap it firmly in place with a strip of elastic adhesive plaster, to which a piece of ordinary plaster is sewed. The plaster passes over the shoulder from the back of the patient to a point near the median line on the front of the chest.

In four or five days, this is removed and any hollow will have been obliterated. Good, firm compression upon the subclavian triangle is important.

One other point about this operation: There is a possibility which the doctor did not mention of injuring the lymphatic duct, especially on the left side. This happened to me in three cases. It has never made the slightest trouble with this obliterating dressing. No drainage was necessary and healing progressed normally. At the same time, I would like to call the possibility to the attention of those who are doing this kind of work.

I think that it is necessary, as Dr. Alexander has pointed out, to wait for a considerable time—months—before one can appraise the value of the operation upon the phrenic nerve. And this is particularly to be noted in the nontuberculous cases.

I learned much from one patient, a young woman, who had a right lower bronchiectasis, with the usual disgusting and alarming symptoms, a great amount of sputum, fetor, etc. I advised resection of the lower lobe, but as a preliminary I performed a phrenicectomy.

Apparently nothing happened. Then I had artificial pneumothorax induced, which simply pushed the diaphragm down—that is all it did—and this form of treatment was discontinued.

Three years afterward she came back under another name. She had been married for two years, and she had had a baby. She was so much improved that nobody would dream of such a thing as performing an operation such as lobectomy. That taught me that it is worth while to wait after phrenicectomy, before deciding upon further operative procedure.

About the paper which Dr. Ballon read, and which to me was extremely interesting of course, I would like to ask Dr. Ballon to say what effect the position of the animal has. Of course, the dog is horizontal and the human posture is vertical.

I approve of the use of the word "worse" in Dr. Alexander's paper. That is a word that ought to be employed oftener than it is. Unimproved doesn't mean much. Worse has a positive meaning which can be safely used in scientific papers, but possibly not in hospital reports. Because of misunderstanding, it might lead to legal complications. And another descriptive word is "convalescent." "Improved," "unimproved," "died" are insufficient in reports of institutions.

DR. CARL A. HEDBLOM (CHICAGO).—Phrenicectomy it seems to me is based on the same principles as other forms of pulmonary collapse and that there can therefore be no more question as to its rationale than concerning that of other methods. As to results Dr. Alexander's presentation seems to me particularly convincing because of the large number of cases he reports and because of the thoroughness and obvious honesty of the study.

As to choice of avenue of approach to the nerve the direct approach after the method of Stuertz seems to me to leave little to be desired from the standpoint of simplicity and of results.

The technic is simple but as we all recognize that latent possibilities of complications are numerous. I have seen the development of Horner's syndrome in two patients; one patient early in my experience on whom I did a phrenicectomy on the worse side, for a bilateral fibrotic tuberculosis died a few weeks later with symptoms of respiratory decompensation. Her respiratory vital capacity was too low for phrenicectomy. One patient had slight transient symptoms of air embolism.

POL. N. CORYLLOS (NEW YORK).—It is with great interest that I have followed the papers of Drs. Decker, Alexander and Ballon, because, as everyone else, I am anxious to come to some definite conclusion as to the precise indications for phrenicectomy and the results we may expect from it.

It is really peculiar that after so many thousands of cases of phrenicectomy published so far, no agreement has been reached on the merits of this procedure, although we have arrived to definite opinions as to pneumothorax and thoracoplasty. On one side we see Sauerbruch in Germany, Berard in France, Dumont and Conbresier in Belgium, Kan in Russia, to mention only a few, who consider phrenicectomy *alone* as of little help in the treatment of pulmonary tuberculosis. In a recent study Max Ernest had only 15 good results out of 185 cases. Matz analyzing the material of the U. S. Veterans' Bureau, found 6.5 per cent good results.

On the other side we have the statistics of Welles with 64.0 per cent "good results," of O'Brien 83.0 per cent "good results" and now the very optimistic results of Dr. Alexander. Dr. Ulmar and I have gone very carefully over 150 cases operated in Seaview and Metropolitan Hospitals within the past 5 years. We found only two cases which have shown any real cure and one of them had a recurrence six months later.

We do not deal here with slight divergences of opinions, but with diametrically opposite assumptions. It is of paramount importance that we arrive at some explanation on that curious condition for the benefit of the profession at large.

It seems to me that we can believe without restriction statistics reporting bad results. We cannot do this with optimistic statistics, especially when the cases have not been followed a sufficient time. On the other hand, with the classification of the National Tuberculosis Association which gives a quantitative but not a qualitative definition of the clinical form of tuberculosis of the reported cases, we do not know whether these cases belonged to the spontaneously resolving or to the ulcerocavitating forms. This is essentially important for the statistics of the surgeons who operate routinely on acute cases, shortly after they are admitted in the sanatorium or the hospital. When one considers that these cases constitute about 20 to 30 per cent of all cases of pulmonary tuberculosis, one can see perhaps the reasons for these discrepancies of opinions and results. During the last two years I have followed the classifications proposed by Ornstein, Dittler and Ulmar. I have come to the conclusion that no surgeon should ignore it because it represents the most rational classification so far proposed.

After 360 thoracoplastic operations, 30 apicolyses and 350 phrenicectomies in which I was guided by that classification I think that I can state that in the statistics of the future the clinical form of tuberculosis should be mentioned for each individual case included in these statistics. In that way we will arrive at some more concrete conclusions about our respective results from phrenicectomies and we will know whether we talk about similar cases or not. The simple diagnosis of tuberculosis is no more sufficient than the generic denomination of "tumors" in the appraisal of our surgical results. There are benign and malignant forms of pulmonary tuberculosis as there are benign and malignant tumors.

Furthermore, it seems to me, that we should eliminate the term "improved" as vague and misleading. The objective of our surgical endeavor is to render the patient bacilli free. Unless and until we have obtained this result we cannot talk about "successful or good results."

I shall close with a word of warning. We have often heard that the results of phrenicectomy are often manifested six or more months after operation. Does this mean that we should wait that long before we decide to perform a thoracoplasty in the cases which phrenicectomy has failed to collapse the cavity? I have seen and we all have seen catastrophes occur because of these long delays. In going over our previous pictures of bilateral cases, how often we have regretted allowing these opportune moments to pass for a more radical procedure. Are we going to give the patient, naturally reluctant to undergo a thoracoplasty, and the not yet fully convinced general practitioner a fallacious pretext for postponement and procrastination?

DR. E. J. O'BRIEN (DETROIT).—I cannot resist taking exception to Dr. Coryllos' remarks. He says 18,000 phrenics have been performed, and he does not know yet whether this procedure can cure pulmonary tuberculosis. I have done over 4,000 myself, and I think I know it can.

Several years ago, I reported over 500 phrenic nerve operations, and the figures are almost identical with those given today by Dr. John Alexander. Approximately 50 per cent of the patients with cavitation and sputum positive for tubercle bacilli became arrested cases. The disease, in most instances, was, of course, minimal.

The trouble is that those who have no confidence in phrenic nerve operations reserve this procedure until the disease has advanced beyond the stage when a phrenic might be expected to accomplish results. They then do a phrenic neurectomy on a patient suitable for a thoracoplasty, get no results, and blame the operation. It is a vicious circle. With proper selection of patients, the results from these operations will be more uniform.

DR. LEO ELOESSER (SAN FRANCISCO).—I would like to ask Dr. Decker, who speaks of a subclavian approach, whether he means a subclavian or a supraclavicular approach?

DR. E. N. PACKARD (SARANAC LAKE).—Dr. Hedblom's report of two fatal cases following phrenicectomy brings up the question whether death may have been hastened by an occlusion of the main bronchus, or smaller bronchi, due to the sudden rise of the diaphragm.

At the present time I have under my care three patients who have had a left-sided phrenicectomy and as a result have a very high rise of the diaphragm. The physical signs and x-ray evidence of atelectasis of the left lung occurred within a week in one of the cases; in another the same condition developed, but at a considerable length of time after the operation. In these two cases the main bronchus is open, but I believe the smaller bronchi are occluded. In the third case all the evidence points to a stenosis of the main stem bronchus.

The first two patients are in fairly good physical condition, but the last patient is acutely ill. Of course there may be no relationship between this condition and the rise of the diaphragm, but I have wondered whether a paralyzed and high diaphragm could cause some of these disagreeable results.

DR. GEORGE HEUER (NEW YORK).—I should like to ask Dr. Alexander a question. It has always been difficult for me to understand why following a phrenicectomy or artificial pneumothorax on one side, there should be marked improvement in the condition on the opposite side.

Dr. Alexander showed in his charts that in cases of bilateral cavitation in which

phrenicectomy was done on one side, the cavity on the opposite improved. Such improvement has frequently been reported by others and there seems to be no doubt of its correctness. I have not been satisfied with the older theories explaining this improvement and would like to ask Dr. Alexander whether he has any new ideas upon the subject.

DR. H. RYERSON DECKER (PITTSBURGH).—I rise only to reply to Dr. Eloesser's question on the approach.

The incision is made on the clavicle. The next step, then, is to bluntly open the fascia just above the clavicle; but it is through the anatomical subclavian triangle.

DR. JOHN ALEXANDER (ANN ARBOR).—Without having studied the patients who are the objects of Dr. Coryllos' pessimistic remarks about phrenic nerve interruption, I cannot attempt to reconcile his "perhaps 5 per cent are permanently cured" with our 34 per cent arrested or apparently cured and 35 per cent improved, quiescent or apparently arrested. The classification of our cases is based upon that of the National Tuberculosis Association. When we used phrenic paralysis for extensive lesions, with large or stiff-walled cavities, we expected and usually obtained only palliation of distressing symptoms, but not arrest of the disease. Our best results were, of course, obtained in patients with only moderately extensive lesions, with or without cavities.

We fully recognize that not a few of these patients would have recovered if no phrenic operation had been performed. There is no intended implication in our presentation that the operation was necessarily solely responsible for the results that occurred. We firmly believe, however, that the results that our patients obtained are far superior to those that unaided bed rest has shown itself capable of producing in a similar group of patients. It is for this reason that we aid bed rest with a phrenic paralysis in those patients whose net chance of recovering has been shown by our own and many others' experiences to be greatly bettered by the operation.

Dr. Eloesser asks the explanation of 100 per cent of deaths in four patients in the last line on one of the tables which the lantern showed incompletely. These were four patients whom we lost track of and we assume that they are dead.

We can offer only a theoretical explanation in attempting to answer Dr. Heuer's question as to how a phrenic paralysis can affect a cavity in the opposite lung. In our published article measurements will be reported that indicate a small shift of the heart away from the side of the phrenic paralysis in a majority of cases. As this slightly lowers the negative pressure in the contralateral hemithorax it affords fibrous tissue in the contralateral lung an increased opportunity of contracting and closing a small cavity. This is likely to occur with the usual small mediastinal shift only if there is a fairly even preoperative balance between the opposing forces of fibrous tissue contraction and negative intrathoracic pressure. It is also probable that the improved resistance against tuberculosis that follows control of the lesions in the worse lung has a favorable influence upon the tuberculous lesions in the opposite lung and indirectly assists in cavity closure.

DR. EVARTS A. GRAHAM (ST. LOUIS).—I shall answer for Dr. Ballon.

Just a word about Dr. Lilienthal's question as to whether results on cough after phrenicectomy on a quadruped would be analogous to those in a biped. I should think that the difficulties of expelling material from the bronchial tree after phrenicectomy would be more extreme in a biped, because of the necessity of pushing against gravity with a cough to a greater extent than would be the case in a quadruped. Therefore, it would seem to me fair to assume that the experimental results obtained on a dog would be less marked than they would be in one who stood in an upright position. So far as the expulsion of material from the lungs is concerned, we are interested not merely in the act of coughing, but the expulsion of material.

BEHAVIOR OF BRONCHOPULMONARY SMOOTH MUSCLE AS DEMONSTRATED BY ELECTROBRONCHOGRAPHIC RECORDS

1. Preliminary Report*

Ethan Flagg Butler, M.D., Elmira, N. Y.

THE existence of bronchopulmonary smooth muscle as well as of pulmonary elastic tissue has long been known. In fact, its gross and minute anatomy has been described in great detail by Baltisberger,[1] Wm. Snow Miller[2] and Macklin.[3, 4] The latter author, in discussing bronchopulmonary smooth muscle before the American Association for Thoracic Surgery in 1929, refers to it as "a motor organ extending uninterruptedly from the larynx to the outermost terminations of the air carrying tubes and infiltrating the entire lung substance." This is an important concept to bear in mind, as it properly stresses not only the continuity, but also the unity of the entire structure.

The physiologic significance of this smooth muscle has not received the same degree of attention. Isolated facts have been proved from time to time. The alternate lengthening and shortening of the bronchi with the respiratory cycle of inspiration and expiration has been clearly demonstrated by Jackson,[5] Francis,[6] Heinbecker[7] and Hudson.[8] The same observers have also shown the changes in caliber and capacity of trachea and bronchi during the respiratory cycle. Bullowa and Gottlieb[9] have advanced the theory of peristaltic action as an aid in the removal of secretions or foreign material from the lower airways. Other functions have been surmised or predicated, and for twenty years there have appeared suggestions that this smooth muscle may aid in the expulsion of air from the lungs during expiration. Its blood supply is derived from the bronchial arteries. It is known to be innervated by the vegetative nervous system. Kuntz[10] states that bronchoconstrictor fibers are supplied by the vagus, and bronchodilator fibers by the sympathetic. Larsell,[11] Kuntz[10] and Pottenger[12] have elaborately discussed the innervation of the lung.

The pathology of the bronchopulmonary smooth muscle has received scant attention. References to structural pathology are apparently limited to the fact that it is damaged to greater or less extent in the degenerative changes in the bronchial walls occurring in bronchiectasis. From the functional standpoint bronchospasm and bronchotetany have

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*From the Chest Clinic, the Arnot-Ogden Memorial Hospital, Elmira, N. Y.

been described; the attacks and symptoms of bronchial asthma have been attributed to dysfunction of this musculature; and Lilienthal[13] has described a lobar atelectasis in thoracic war injuries that might well be due to muscle spasm.

Such, in general, was the state of knowledge at the close of the last decade. Since that time, however, extremely important observations have been reported by Luisada.[14, 15, 16, 17, 18, 19, 20, 21] His methods, findings and conclusions have added an important chapter to the physiology of respiration, and have laid a solid foundation for the further investigation of the functional pathology of bronchopulmonary smooth muscle. It is desirable to briefly review his work and to mention, as nearly as possible in his own words, one dramatic incident which spurred him on to further investigation. An experiment in pulmonary oncometry had been finished, the animal was dead, one lung remained within the oncometer, which was still attached to a tambour and recording apparatus. Twenty minutes after the death of the animal, and without any further stimulation, there was suddenly written on the kymograph the record of spontaneous, rhythmic variations in lung volume. This could not result from any phenomena associated with elastic tissue. It must result from some property of the smooth muscle itself.

Luisada realized that whenever muscular contraction occurred there must of necessity be a change in electrical potential, therefore, he devised the new and important method of recording the action currents of bronchopulmonary smooth muscle. To these records he gave the name "electrobronchogram," which may be, for convenience, abbreviated to "EBG." The method itself he termed "electrobronchography." These terms are directly comparable to "electrocardiogram," "EKG" and "electrocardiography," and they imply exactly the same manner of study; the one of bronchopulmonary smooth muscle, the other of cardiac muscle. Before a perfected technic could be developed, certain difficulties had to be overcome. Chief among these difficulties was the elimination of the omnipresent "EKG." To accomplish this, close contact had to be secured between the two electrodes and the relatively weak muscle whose record was desired. In the intact living animal and in the human subject this could only be accomplished by the introduction of an endobronchial electrode, insulated except for its distal tip, into a small branch bronchus, preferably in the right lower lobe. This was, by choice, the negative electrode. The positive electrode was either fastened directly to the trachea of tracheotomized experimental animals or fastened about the neck of the human subject, in the same manner that an electrocardiograph cuff is fastened about an extremity. Both electrodes should be moistened with normal saline solution. Even with such close contact, the cardiac currents tended to overshadow the desired currents until Pasoli[22] devised a grid tube filter which he designated a thermoionic amplifier-filter. This served to select for amplification the action cur-

rents arising in the bronchopulmonary smooth muscle, while, at the same time, it served to selectively diminish the intensity of the cardiac action currents, before their arrival at the recording apparatus. Any standard electrocardiograph might serve as recording apparatus.

Another difficulty lay in coordinating the action current with a definite phase of the respiratory cycle This was accomplished by securing a simultaneous record of either abdominal respiration or the pneumotachographic curve (P.T.G.). In either case the curve denoting the respiratory phase could be recorded upon the same film by a separate device activated not by the galvanometer, but by pressure changes in a tambour.

Having overcome the mechanical and technical difficulties, Luisada was able to carry out a lengthy series of investigations which led to certain important deductions as to the physiology of this musculature. They are:

1. That bronchopulmonary smooth muscle is truly a motor organ which, by its contraction during expiration, actively assists in expelling air from the lung.

2. That in normal respiration there is a pulmonary systole and diastole: the former occurring during expiration, the latter during inspiration.

3. That bronchopulmonary smooth muscle is in close reflex relationship with the nasal and pharyngeal mucosa.

4. That the amplitude of the EBG curve and, therefore, the contractility of bronchopulmonary smooth muscle, varies directly with the amplitude of respiration, but indirectly with the rate of respiration.

5. That the amplitude of the EBG curve increases as a result of vagal stimulation and diminishes as a result of vagal inhibition or sympathetic stimulation.

6. That to some degree the normal behavior of the bronchopulmonary smooth muscle is dependent upon the normal impulses traveling through the phrenic nerve, as there is a diminution of EBG amplitude following section or avulsion of the phrenic nerve.

Luisada also arrived at the following deductions as to the functional pathology of the bronchopulmonary smooth muscle:

7. In asthmatic attacks and in histamine poisoning there is a complete dissociation of the normal reciprocal relations between striated body muscle and pulmonary smooth muscle in the respiratory cycle. No longer does one contract during inspiration and the other only during expiration, but the contraction of pulmonary smooth muscle commences before the inspiratory act is complete and not only becomes antagonistic to the motor mechanism of inspiration, but also fails to render aid during expiration.

8. With the exception of morphine in small doses and ether by rectum, all the general anesthetic agents and narcotics investigated by Luisada caused a diminution in the amplitude of the EBG curve.

9. Reflex relationship can be demonstrated between stomach and lung and between coeliac plexus and lung. These reflexes can be abolished by spinal anesthesia or splanchnic block.

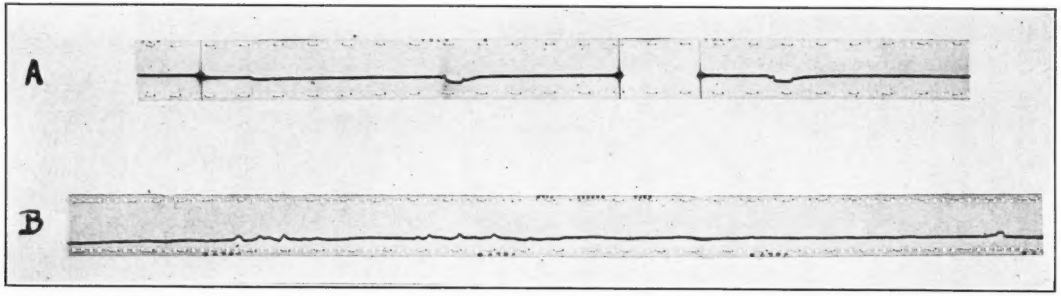

Fig. 1.—*A.* Electrobronchographic record of spontaneous pulmonary contractions in cow's lungs removed at slaughter house and isolated from rest of body.

B. EBG of spontaneous pulmonary contractions in lungs remaining in situ within a widely open thoracic cavity of recently sacrificed rabbit.

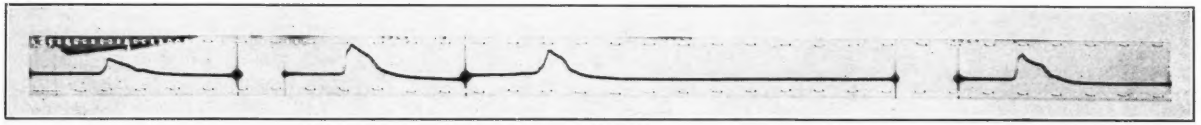

Fig. 2.—EBG obtained in response to mechanical stimulation of vagus in isolated cow's lungs removed at slaughter house.

Matas, in discussing postoperative pneumopathies, has already called attention to Luisada's work. Electrobronchography is so important, however, that it deserves individual and specific mention before a group of men who are particularly interested in thoracic problems. It should prove of great value, not only in investigating normal respiratory physiology but also such obscure problems in pulmonary functional pathology as asthma, allergy, atelectasis and the postoperative pneumopathies. It has already been suggested by Luisada,[20] Matas,[23] Pottenger[24] and the author[25] that some of these pneumopathies may result from viscero-visceral reflexes manifested in the bronchopulmonary smooth muscle.

With the object of laying the foundation for more elaborate studies, investigations were initiated in Elmira in 1932, and have progressed to a point that warrants a preliminary report on the elemental aspects of the subject.

SPONTANEOUS PULMONARY CONTRACTION

Luisada found this to be of relatively rare occurrence. It can only be accepted when it occurs in lungs isolated from the rest of the body, and in the absence of any vagal stimulation. It is more apt to occur in lungs that have been previously inflated to a moderate degree. That it does occur has been proved by Luisada and is corroborated by the following observations:

1. Experiment No. 4, Oct. 3, 1932. Both lungs were removed from a freshly slaughtered cow. With the material still warm and moist, the positive electrode was introduced into the depths of the right lower lobe, the negative electrode was fastened to the severed end of the trachea. Action currents were obtained satisfactorily in response to inflation and deflation through an airtight cannula and in response to vagal stimulation. The lung was then allowed to rest and at irregular intervals and without either vagal or mechanical stimulation, minimal variations in potential were observed, but no gross evidence of pulmonary contraction. This is shown in Fig. 1-A.

2. Experiment No. 7, April 9, 1932. White female rabbit, weighing 2.63 kg., anesthetized with intraperitoneal morphine sulphate. After tracheotomy the normal EBG and other observations were recorded. After the animal was sacrificed and the chest widely opened, the lungs were allowed to remain undisturbed, but still connected, airtight to a closed system wherein pressure was left at +3.5 cm. water. Pressure variations from 0.5 cm. to 0.1 cm. occurred from time to time. The EBG of some of the smaller contractions are shown in Fig. 1-B.

RESPONSE TO VAGAL STIMULATION

The vagus, according to all experimental evidence, contains motor fibers to bronchopulmonary smooth muscle. Mechanical stimulation of the vagus in the isolated cow's lungs used in experiment No. 4 produced definite smooth muscle action currents as shown in Fig. 2.

RESPONSE TO INTRAPULMONARY PRESSURE

Luisada came to the conclusion that the normal stimulus giving rise to bronchopulmonary smooth muscle contraction was the distention of

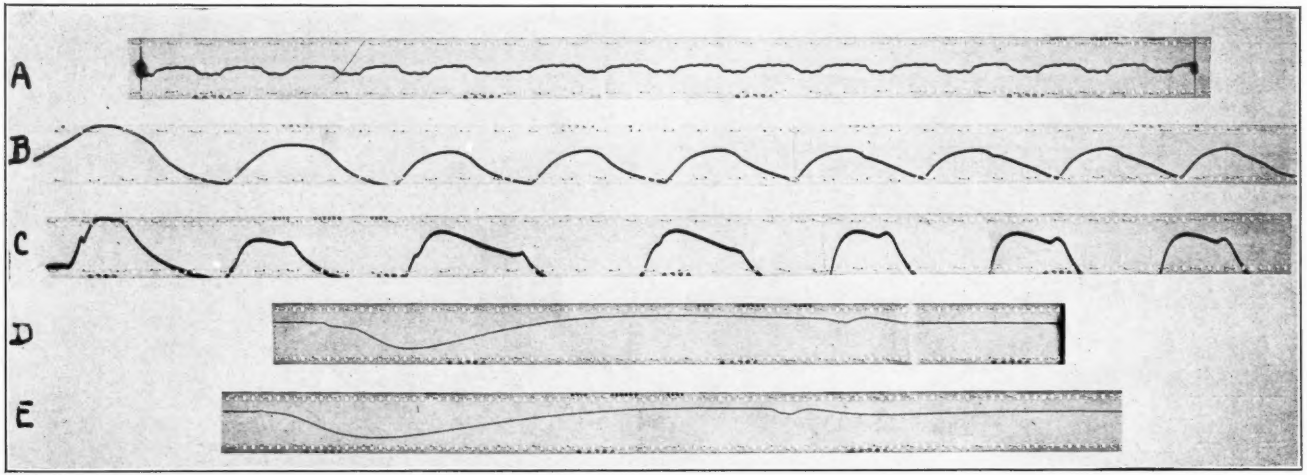

Fig. 3.—*A*. EBG of normal respiration in tracheotomized rabbit.
B. EBG obtained in response to rapid mechanical inflation and deflation of isolated heifer lungs removed at slaughter house.
C. EBG obtained in response to rapid mechanical inflation and deflation of human lungs removed at autopsy.
D and *E*. EBG obtained in response to slow mechanical inflation and deflation of rabbit lungs remaining in situ within closed thorax after sacrifice of the animal.

the lungs at the end of expiration. This can be simulated experimentally by mechanical inflation and deflation of isolated lungs or by mechanical inflation and deflation of lungs within the closed thorax of the dead animal. Whether the lungs are isolated from vagal control or not, the curve remains the same and closely resembles the normal electrobronchogram. It is, therefore, fair to assume that under normal conditions distention or stretching of the smooth muscle fibers acts as the normal stimulus leading to their contraction. The following experimental evidence supports this view:

Experiment No. 7, April 9, 1932. White female rabbit anesthetized with intraperitoneal morphine sulphate. The electrobronchogram of normal respiration through a tracheotomy tube is shown in Fig. 3-A.

Experiment No. 4, October 3, 1932. The lungs of a freshly killed heifer were completely removed from the carcass and alternately inflated and deflated through an airtight cannula fitted into the trachea. The action currents obtained in response to this stimulus are shown in Fig. 3-B.

Experiment No. 3, Sept. 19, 1932. Human lungs removed at autopsy, while still moist and warm, were inflated and deflated through an airtight cannula. The action currents obtained are shown in Fig. 3-C.

Experiment No. 6, March 19, 1933. White male rabbit, weight 2.2 kg. After a series of experimental observations, the animal was sacrificed. With thorax closed and lungs in situ, inflation and deflation gave the curves shown in Figs. 3-D and 3-E.

RESPONSE TO EXTERNAL COMPRESSION

Under unusual conditions not encountered in ordinary quiet respiration, there is apparently an additional stimulus to smooth muscle contraction consisting of compression of pulmonary tissue. It is possible to recognize a characteristic action current picture in response to pressure exerted upon the external surface of the lung, whether the lung be isolated or whether the lung be within an intact and living animal. This must be recognized as a definite response of bronchopulmonary smooth muscle to abnormal stimulation. Experimental evidence supporting this statement is noted below:

Experiment No. 3, Sept. 19, 1932. Freshly removed, warm, moist human lungs were obtained at autopsy. The patient had died of pneumonia. The right lung was free of disease, the left lung was pneumonic. With airways open; manual compression of the right or comparatively normal lung gave rise to action currents shown in Fig. 4-A. Manual compression of the left lung gave rise to action currents shown in Fig. 4-B. These curves differ and lead to interesting speculation as to the effect of pulmonary disease on the smooth muscle.

Experiment No. 4, Oct. 3, 1932. Isolated lungs removed from a freshly slaughtered heifer immediately after death. Lungs were moderately distended by an airtight cannula introduced into the bronchus. The beam of the recording apparatus was allowed to come to rest, then the lungs were manually compressed, with resulting action currents as shown in Fig. 4-C.

Experiment No. 6, March 19, 1933. White male rabbit, weight 2.2 kg. Tracheotomy had been performed. The effect of atropine had been studied. After

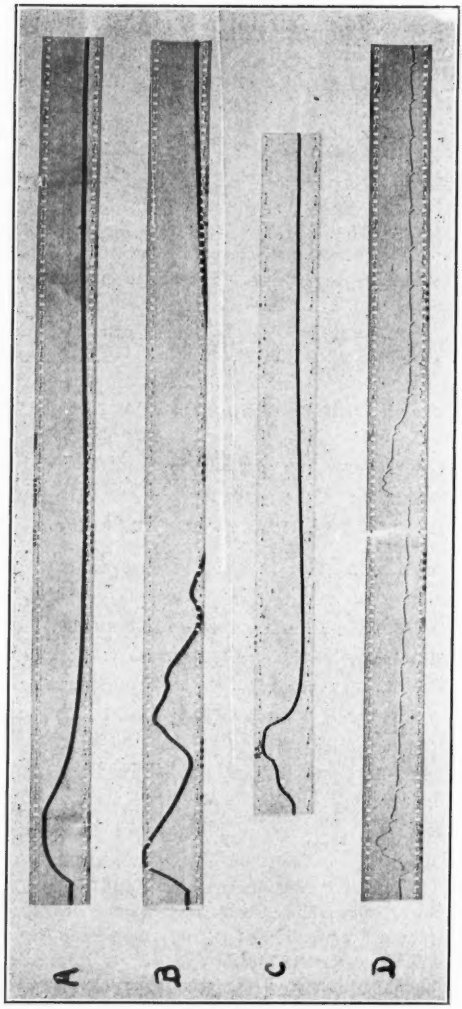

Fig. 4.—*A.* EBG obtained in response to external compression of normal human lungs removed at autopsy.
B. EBG obtained in response to external compression of pneumonic human lungs removed at autopsy.
C. EBG obtained in response to external compression of heifer lungs removed at slaughter house.
D. EBG obtained on living rabbit, thorax closed, after tracheotomy tube had been completely occluded. The EBG is not synchronous with respiratory efforts, which were violent but unaccompanied by any pulmonary ventilation.

two hours of observation, the tracheotomy tube was completely occluded. Immediately violent muscular respiratory efforts were apparent with no change whatever in the air content of the lung. The respiratory rate had ranged from 35-45 per minute up to this time. In the absence of to and fro passage of air through the airways, there were obtained curves quite similar to those observed as a result of external compression of lung and, furthermore, neither the latent period nor the reaction time coincided with the respiratory efforts, but took on a rhythm of less than 8 per minute quite independent of body muscle contraction. These curves are shown in Fig. 4-D.

CONCLUSIONS

An important new method for investigating bronchopulmonary smooth muscle, electrobronchography, has been developed by the Italian investigator Luisada.

Pulmonary systole and diastole has been proved to be a factor in the normal mechanism of respiration, the systole occurring during expiration and aiding in the expulsion of air from the lungs.

Stimulation of the vagus causes contraction of bronchopulmonary smooth muscle.

The normal stimulus to contraction of bronchopulmonary smooth muscle is distention of the lung by inspiration.

An additional stimulus, not encountered under normal conditions, is external compression of lung tissue.

Spontaneous contractions may occur in isolated lungs without any external stimulation.

I wish to express my gratitude to Mr. Galen Clark of the Victor Electric Company and to Miss Norma M. Lay for valuable technical aid in the conduct of these experiments.

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DISCUSSION

DR. POL. N. CORYLLOS, (NEW YORK).—The methods of accurate exploration and of recording the action of the smooth musculature of the lung, developed by A. Luisada in the service of Professor Frogoui, of the Medical School of Padua, Italy, is of special interest to me, because of its potentialities in the minute investigation of pathogenesis of atelectasis.

The removal of the electrocardiograms from the graph and the development of the electrobronchograms, by means of the thermoionic valve of Pasoli has rendered it possible, for the first time, to follow the changes produced upon the smooth bronchial muscles by a number of mechanical, biological pharmaceutical or infectious changes of the lung.

Luisada has shown so far that normally there is a perfect synergy between the striated respiratory muscles and the smooth bronchial muscles. During inspiration the bronchial muscles are relaxed, whereas they contract during expiration. Thus the lung is not simply a passively elastic organ but also an actively contracting one. Expiration becomes in that way a passive phenomenon for the chest but an active phenomenon for the lung.

Furthermore the peristaltic bronchial motions, the existence of which had been shown by Reinberg, Macklin and Hudson, are of the greatest importance as a means of defense of the lung. It will be of interest to investigate what happens to these motions in bronchial obstruction. It is very probable that peristalsis will be increased as in the obstruction of the intestine which is embryologically homologous to the bronchi and it has a similar dual innervation, vagal and sympathetic.

I have the impression that the similarity of innervation of these two organs goes even further. In investigating with Dr. Birnbaum upon the effect of complete denervation of one lung in the dog by resection of the vagosympathetic, middle cervical and stellalum ganglia, and of the first four dorsal sympathetic ganglia and rami, we found that no changes were produced in the lung. Atelectasis never occurred spontaneously, thus proving that the result obtained by Fontaine and Herman was due to an error of interpretation. Obstruction of a bronchus or infection with pneumococcus did produce atelectasis or lobar pneumonia exactly as in the intact lung. It appears therefore that, as in the intestine, the finer regulation of the bronchial muscles is under command of an autonomous pulmonary plexus. Future investigations may show what action the efferent and afferent nerve fibers have upon it and whether Dixon and Brodie are right or not.

The possibility of taking electrobronchograms on the human will perhaps help to advance our knowledge upon the action of the bronchial muscles during asthmatic attacks. The fact brought up by Luisada, that in anaphylactic shock, in animals, there is dissociation of the movements of the striated respiratory and of the smooth bronchial muscles and disturbance of their synergetic action, seems to open a new horizon in the study of the asthmatic manifestation.

I want to congratulate Dr. Butler upon having attracted our attention to the work of Luisada and having communicated to us his own results.

DR. HOWARD LILIENTHAL (NEW YORK CITY).—I am interested in this particular study, and would call attention to the fact which Dr. Butler was kind enough to mention, that in a report on Operating Team No. 39 during the War—published in the March 22 number of the A. M. A. Journal 1919—I made the suggestion that the so-called traumatic atelectasis which we saw in the open chest was produced by spasm.

Now Dr. Butler has shown the action of the muscles which Macklin showed us existed in the ultimate parts of the lung the existence of which had been previously denied. I have seen lobar atelectasis occur almost instantaneously in the open chest during operation.

PNEUMONECTOMY FOR SARCOMA OF THE LUNG IN A TUBERCULOUS PATIENT*

HOWARD LILIENTHAL, M.D., F.A.C.S.
NEW YORK CITY

MY REASON for presenting the following case history is not merely because of its great scientific importance nor that it contributes new methods, nor exemplifies an unusual success—indeed its termination is a fatal one. I am bringing it before this association in the hope that it will arouse discussion.

The points before us are: First, when may clinically cured pulmonary tuberculosis cease to be regarded as a specific risk in thoracic surgery? Second, what choice of treatment have we, operative or not, in tumors of the lung in patients with tuberculosis of the tumor-bearing lung or its fellow? The two questions are so intimately related that I have not found it possible to discuss them separately.

CASE HISTORY

Miss E. K., a woman of thirty-four and of normal appearance, was referred to me by Dr. J. Burns Amberson, Jr., on January 19, 1933. She had had pulmonary tuberculosis occupying most of the upper part of both lungs. Her illness had been diagnosed eleven years before, in 1922, after a hemoptysis, and then for a year and a half she had been treated at Loomis Sanatorium. A brief résumé by Dr. Amberson is as follows:

"The upper half of the right lung was involved in a recent exudative tuberculosis and just beneath the clavicle there was a cavity 3½ cm. in diameter. This lesion tended to resolve and the cavity gradually contracted and became obliterated. However, before this was accomplished there was a small bronchogenic spread into the middle third of the left lung in December, 1922. This also underwent resolution and in a few years calcification appeared in both lungs."

On her discharge the case was considered one of clinical cure or complete arrest. For the following ten years she had lived a normal life, or until March, 1932, when there was a slight elevation of temperature without cough or other signs of phthisis. An x-ray film then revealed a small opacity in the left upper lobe. (Fig. 1.) This was interpreted as probably a tuberculous lesion, but it increased in size rather rapidly without the customary general signs of the disease and when I first saw her eight months later there was a circular opacity about the size of a small orange extending downward to the interlobar fissure and inward to the mediastinum. There were no other suspicious areas in either lung except those representing the scars of her old disease. (Fig. 2.)

She entered Mount Sinai Hospital a week after her first visit to me and the

*Read at the Sixteenth Annual Meeting of the American Association for Thoracic Surgery at Washington, D. C., May 9, 10, 11, 1933.

following day raised for the first and only time a tiny mass of bright bloody sputum. She looked the picture of health in nutrition and in color. Her blood pressure was 120/85; pulse 78 and excellent in quality; respirations 20; temperature normal.

Fig. 1.—Tumor of left upper lobe strongly suggesting tuberculosis—August 8, 1932.

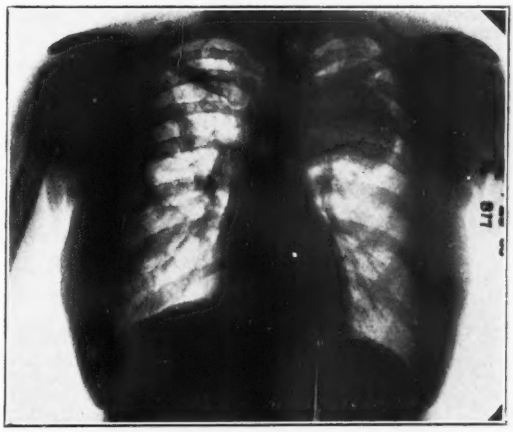

Fig. 2.—Same case as Fig. 1, but five and one-half months later; now, clearly, a neoplasm.

The diagnosis of tumor rested mainly on the rapidly increasing area of the shadow in the film. Dr. Jaches and Dr. Wessler were of the opinion that the opacity probably represented a primary malignant tumor of the left upper lobe, at first not near the mediastinum but now probably adherent to it. After conference with Dr. Amberson I decided that since this patient had no chance for recovery without

surgery, an operative attempt should be made and the procedure concluded according to the conditions which would then be disclosed. In coming to this decision we took into consideration the history from the time of entrance into Loomis Sanatorium on April 18, 1922. There were then the characteristic signs of tuberculosis, with temperature to 103° F. by mouth, continuing with variations for a few months. The sputum was positive. The weight was 2 per cent under normal. The thoracic mobility was restricted, especially on the right. There is a summary note of "moderate infiltrations scattered through the upper two-thirds of the right lung, apparently most intense about the hilum; probably slight apical and perihilar infiltrations on the left. Far advanced B." The heart was normal and the blood

Fig. 3.—Urinary tract outlined with uroselectan. (Swick.) Note irregularity of bladder produced by a tilted uterus.

pressure was 116/64. The sputum had been positive for thirteen months after her admission to Loomis Sanatorium when it became *negative and all expectoration ceased.**

Four years after her discharge from Loomis Sanatorium the late Dr. Bertram Waters examined Miss K., and gained the impression that continued retrogression with fibrosis was present in the right lung and that there was no acute left-sided lesion. The x-ray pictures made at this time revealed old calcifications in the right side with fibrotic infiltration about the upper horn of the hilum. There was largely fibrotic infiltration at the apex with some calcified nodules behind the clavicle and a few deposits in the base.

*Notes furnished by Dr. George Foster Herben, Physician-in-Chief of Loomis Sanatorium.

On the left there was moderate fibrotic infiltration between the second and fourth ribs laterally and slight deviation of the trachea to the right.

The patient was kept under close observation and it was not until May 28, 1932, that the note was made of the rounded area of uniform density 4 by 4 cm. in the area adjacent to the upper pole of the hilum, the medial border sharp and the outer somewhat fuzzy. This opacity was interpreted as being probably of a tuberculous nature, although its characteristics were somewhat atypical.*

Following this there was a slight increase in the size of the shadow and on May 25, 1932, Dr. B. T McMahon of Loomis Sanatorium stated that "Miss K. developed some elevation of temperature—about 101.2° by mouth—which subsided in three days." There was, however, no loss of weight and her fever was supposed to have been, possibly, the result of influenza. Throughout the ten-year period she had been working full time in a normal fashion. The blood pressure had become elevated to 152/80. Still the shadow had been regarded as a manifestation of tuberculosis and one could scarcely have been blamed for this in the face of the early history.

She was put to bed and on Aug. 11, 1932, Dr. Herben found that the "new area over the left lung" was more sharply circumscribed, "smaller, and considerably less dense."

In order that no diagnostic procedure should be omitted Dr. Amberson advised roentgenologic examination of the kidney regions to determine the presence or absence of a shadow which might be cast by a hypernephroma. After the administration of uroselectan intravenously by Dr. Swick, nothing abnormal was found except a malposition of the uterus contrasting with the bladder filled with the opaque medium. (Fig. 3.) On gynecologic examination there was no enlargement of that organ and no tender areas; merely flexion to the right.

Endoscopy, always essential in cases of this kind, was deferred until the time of operation on January 28, 1933, when Dr. Rudolph Kramer passed the bronchoscope, finding the trachea and bronchi absolutely normal.

Dr. Amberson, Dr. Douglass of Loomis Sanatorium, and Dr. Jacob Goldberg, formerly of Loomis Sanatorium and who had treated the patient, were present.

OPERATIVE PROCEDURE

With the bipolar electric scalpel a long sixth interspace incision was made and its posterior extremity continued upward. The fourth, fifth, and sixth ribs were divided near the spine and the rib retractor was inserted, perfectly exposing the field of operation. There were a few cobwebby adhesions between the upper lobe and the axillary part of the chest wall but none on the mesial side, so the lobe could be easily drawn into the wound and the solid tumor exposed.

I had intended to remove the upper lobe only, but this proved to be impossible on account of the relation of the tumor-bearing portion with the lower lobe bronchus. So with the approval of Dr. Amberson the entire left lung was ablated after the pedicle had been crushed and ligated in sections with heavy chromicized catgut sutures. Because of the size of the neoplasm I was obliged to leave the stump rather short. The lumina of the bronchi were painted with pure phenol. The ligatures were tied together and sutured to the soft parts of the wall so as to steady the mediastinum. The phrenic nerve as it ran over the pericardium was firmly crushed and the diaphragm paralyzed. A section of about 1½ inches was removed from the sixth rib, leaving an opening from which the ligatures emerged, and they were held to the chest wall by a transfixing safety pin. A drainage opening was now made in the ninth interspace and a tube with a flapper valve made with a rubber finger cot was inserted air tight. Bleeding had not been alarming and the vessels in the chest wall were secured by electrocoagulation, a few by ligation.

*This was not a Loomis film.

The ribs were brought together with a single large pericostal chromic catgut suture, which approximated the edges perfectly. A few interrupted sutures of catgut were passed through the soft parts and silkworm-gut stitches united the edges of the skin.

At the conclusion of the operation the patient's condition was good, the pulse regular, and the breathing quiet. At Dr. Branower's suggestion because of the large and sudden loss of lung tissue, a permanent oxygen-bearing catheter was placed in the right nostril.*

There was but little shock after this operation and twenty-four hours later the oxygen was discontinued. The respirations then, without the oxygen, were between 20 and 22 and were not labored. The pulse was of excellent quality and 120 in rate.

In forty-eight hours the temperature rose to 102° F.; pulse 120, but the respirations still continued at from 20 to 22 per minute. The valve functioned well and about eight ounces of bloody serum had drained since the operation. The blood pressure was 104/54.

Then there began a hacking but distressing cough without expectoration. Pain was almost absent and morphine was given because of discomfort only. The cough continued and there were tracheal râles. The temperature rose to 104° F. but then receded. The pulse remained excellent in quality and the blood pressure maintained its systolic reading of between 100 and 110.

Four days after operation the respiratory distress had become unbearable and I requested the resident laryngologist, Dr. Max Som, to make an attempt to empty the trachea by suction. Up to this time there had been no outstanding signs of solidification in the right chest. Some rather purulent material was removed and there was temporary relief and, although the performance was strenuous and shocking, the patient requested a repetition of the suction.

The afternoon of the same day I took her to the operating room again where with the help of nitrous oxide and oxygen anesthesia I reopened the entire wound, hoping that by removing the bronchial ligatures some good might be accomplished. There was not the slightest sign of infection and the chest looked about as it did at the time of operation. I removed the ligatures but there was only the faintest hissing from the stump, the bronchial walls being apparently adherent. I sutured the wound completely during intrapharyngeal hyperpressure. The drainage tube was tied so that there would be no motion of the mediastinum. Dr. Kramer then passed the bronchoscope and removed a little pus from the right bronchus, but no mucus. Three hours later the patient died.

Permission was given for postmortem examination through the large wound and Dr. Paul Klemperer reports about as follows:

ABSTRACTS FROM POSTMORTEM EXAMINATION

(Furnished by Dr. Paul Klemperer, Pathologist to Mount Sinai Hospital.)

There was no edema, cyanosis, or petechiae. Nothing to indicate deflection of the mediastinum. The large pulmonary vessels were securely tied as well as the left bronchi.†

A few large parabronchial lymph nodes were found. The apex of this lung was covered with short depressed scars. The interlobar fissure between the right

*It is the suddenness of the withdrawal of lung space and not its mere volume which is of importance. Life can be maintained with less than one-fifth of normal aeration.

†I thought that I had removed all ligatures from the stump but evidently this had not been complete.

upper and middle lobes was obliterated by adhesions. The right lower lobe was bluish red and distended; it was nonrepitant except for a small portion at its apex. Irregular nodules were present and the surface of the sectioned lung was deep red in which were raised gray patches measuring from a few millimeters to one centimeter in diameter. At the apex of the lower lobe there was a patch of gray areas the size of millet seeds, apparently tuberculous. Considerable turbid edema-fluid ran from the cut section.

The middle lobe contained a few stony crepitating bodies which were a few millimeters in size. There was a well-encapsulated cavity the size of an olive, lined with smooth walls, and containing dirty greenish exudate and yellow caseous material. This cavity did not connect with the bronchi; it rested immediately on the interlobar fissure four centimeters from the apex of the middle lobe, but had not broken into the fissure.

The trachea and bronchi were congested, but the mucosa was intact.

A smear of the lung revealed gram-positive cocci in clumps and diplococcus forms. There were no tubercle bacilli.

The report on the left lung, the one which had been removed, brought out a number of important facts. The tumor in the left upper lobe was eight centimeters in its greatest length, parallel with the long axis of the lobe, and was four and one-half centimeters in its greatest width. The bronchi, as had been gathered from Dr. Kramer's endoscopic examination, were not at all involved in the growth nor did they show any abnormality except the separation of their branches where these were pushed aside by the tumor. The mass had a distinct capsule from one to five millimeters in thickness composed of firm almost cartilaginous translucent tissue elevated sharply from the surrounding lung. In the pleural surface of the lung there were a few scars representing old tuberculosis with calcification and one area with ossification, and there was also some brown infiltration. In this part of the lung as might have been expected there was moderate atelectasis. Over a space of about ten millimeters a nodule projected outside of the lung and was also surrounded by a cartilaginous capsule. The interior of the tumor was fleshy white opaque tissue, very friable and arranged in smaller and larger nodules from one to three centimeters in diameter and separated by softer areas of necrosis with partial cavitation. (Fig. 4.) This necrotic tissue had a peculiar tan color and was associated with some hemorrhagic areas. None of the pulmonary scars were in continuity with the tumor. The very close proximity to the hilum noted in the x-ray films was confirmed but the lymph nodes in this region were not abnormal.

"The left lower lobe shows no abnormality."

The pathologic diagnosis was "metastatic round-celled sarcoma of immature type. Calcified tuberculosis and recent foci of tuberculous bronchitis and bronchopneumonia." The microscopic description follows:

"Section through tumor mass reveals nodular masses of tumor cells with well-defined capsule. The cell type is a large cell of varying shape for the most part spherical with a large nucleus, pale staining chromatin network, and nucleolus. There is a fine reticular framework. The cells grow in spherical masses, in places replaced by a golden yellow pigment and through which thin-walled distended vessels run. The capsule is infiltrated. Neighboring lymph node shows no tumor cells. No tubercles.

"Section through bronchus containing plug of necrotic fibrinous tissue with remains of round cells and polymorphs. On one side of the bronchus alveolar cells and foreign body giant cells. Beyond this, and on the other side of the bronchial wall tumor tissue is seen. Tbc. stain—positive for tubercle bacilli.

"Section shows tubercles with large calcified centers, in one of which numerous giant cells of Langerhan's type are seen. Nearby, there is a more recent tubercle,

with beginning caseation in center, numerous epithelioid cells, and a wide ring of lymphoid infiltration—alveoli are filled with lymphoid cells and alveolar cells bound together by fibrin. This is, in places, the seat of necrosis, in other places, that of organization. Other areas show lung alveoli containing a few round alveolar cells —and at the periphery lung alveoli are distended and walls ruptured. Positive for tubercle bacilli.''

Fig. 4.—Section of upper left lobe, showing the sarcoma and its relations to vessels and bronchi.

SECTION THROUGH THE RIGHT LUNG

''Section through the right lower lobe at the base reveals a thickened congested pleura. Not an intact bronchus remains, the lining epithelium is desquamated and the wall is the seat of a serohemorrhagic exudative process; in places the site of a bronchial lumen is occupied by a plug of polymorphonuclear cells and neighboring alveoli are the seat of a polymorphonuclear pneumonic exudate. The entire section

reveals edema and congestion, distorting lung parenchyma—areas that are not the seat of polymorphonuclear pneumonia contain serosanguineous exudate with many dark round alveolar cells. Megakaryocytes are very numerous and occasional giant cells are seen.'' Tbc. stain—positive but scarce.

''Section through right lower lobe at apex reveals a congested pleura, subpleural emphysematous areas, beyond this congestion, edema, and areas of extensive bronchopneumonia; the exudate is of a polymorphonuclear nature, the bronchi show destruction but elastica stain reveals good maintenance of alveolar framework. Megakaryocytes and giant cells are seen. Tbc. stain—positive but scarce.

Fig. 5.—Uterus, showing primary intramural sarcoma.

''Section through wall of cavity in right upper lobe reveals a lining of necrotic tissue with occasional areas of granulation and fibrous-tissue formation. Spaces occupied by cholesterol crystals may be seen. At one place the wall is severely congested and contains large, thick-walled distended vessels. Lying deeper in the wall of the cavity are numerous large tubercles with caseating centers and calcium deposits with a capsule of fibrous and granulation tissue. One large bronchus in the section is plugged with epithelial débris, has a partially desquamating lining and smaller radicles of the bronchial tree are filled with large round alveolar cells.

''Section through parabronchial lymph node reveals neither tuberculous nor neoplastic tissue.''

GENITAL SYSTEM

The uterus was small and firm. Incision along the posterior aspect revealed a slightly enlarged uterine cavity. The endometrium was congested and somewhat hemorrhagic but with no suggestion of tumor. There was a thin tab-like pedunculated polyp arising from the internal os evidently fibroadenomatous. A firm, plum-sized, well-delimited mass was present in the anterior wall of the uterus and incision revealed a tumor, the lobules ranging from one-half to one and one-half centimeters in diameter, gray in color and soft and very cellular in appearance. One portion of this mass was hemorrhagic and there were several small areas filled with a grumous yellow material. Nothing else of importance was found. (Fig. 5.)

MICROSCOPIC EXAMINATION

"Sections through the uterine tumor mass showed it to consist of islands of cellular structure with fibromuscular tissue intervening (van Gieson). The cells vary in shape from spheres to elongated forms and range from the dimensions of a red blood cell to four times this size. The nuclei are large, contain a fine chromatin network and one or two nucleoli. Mitotic figures are few. There is a fine fibrous-tissue network interlacing between the cells (van Gieson). The cells grow in strands and whorls and invade the interspersed fibromuscular tissue. Small thin-walled vessels run through the neoplastic tissue and the cells are arranged in radiating fashion along these capillaries. Some of the lobules show congestion, others necrosis and in places calcification and deposits of yellow pigment.

"The neighboring uterine musculature is, in itself, intact, but many of the vessels contain nests of neoplastic tissue within the lumina; some thrombus formation.

"Section through the uterine and cervical mucosa shows a moderate degree of congestion with some hyperplasia of the uterine glands but no neoplastic tissue."

DIAGNOSIS

"Sarcoma of uterus (intramural, anterior wall).
"Chronic fibrocaseous tuberculosis R.U.L., with healing cavity.
"Pulmonary edema.
"Metastatic sarcoma of left upper lobe."

It is clear from the description of the left lung that in spite of the absence of sputum this patient still had areas which must be recognized as open tuberculosis, but it has long been realized that mere absence of sputum in so-called clinical cure is no absolute guarantee that a person may not have microscopic open tuberculosis. There is, however, no reason for taking this into special account in operations upon the lung of a former tuberculous patient. In the case of one who has been "cured" of the disease and who may require, as did Miss K., an operation not related to the old process, I would stress this point particularly, because the disease which threatens life is the one which is to be attacked.

Pulmonary tuberculosis is so nearly universal that to regard the existence of a hitherto unsuspected case, healed or otherwise, as a contraindication for operation upon the thorax would practically eliminate all surgery of the chest. Still, we must take the presence of even mildly

active lesions into account when deciding upon an operation upon the lung itself. Manifestly, dealing with a disorder in another part of the body, the danger must be weighed against that of the pulmonary lesion. Since this history is one of a fatality it is but fair to consider what other course might have been open to me.

It seems clear that a diagnosis which carries with it a reasonably sure prognosis, good or bad, is essential but when this is not possible without placing the patient in jeopardy, it is out of the question. For example, tumors of the lung without metastasis require open thoracotomy for diagnosis. We may steal a specimen from the tumor with the aid of a punch if there are firm adhesions to the parietes; and if the skin incision is made with the electric knife, the dangers of disseminating the neoplasm are negligible. The probability of hemorrhage is not as great as from a needle puncture.[1] It is hoped that in the future other methods may be developed.

In the case in question when there was no certain way of estimating the character of this rapidly enlarging neoplasm what should have been done? What would I do if I were again confronted with a similar condition?

Blind treatment with radiation of one or another kind seems hardly fair to the patient. It must be remembered that there was no discoverable connection with the bronchial system, certainly not one which could be reached by endoscopy for the removal of a specimen. All accessible parts of this patient's anatomy were explored by such means as were at our command and the Kahn test for syphilis was proved to be negative.

Had I suspected that this case was one of sarcoma, a rare tumor in this location and a metastasis at that, I would almost certainly have advised treatment by Coley's mixed toxins.

With a single large increasing neoplasm, possibly operable, I still believe that an attempt to extirpate it would be not only justifiable but correct. For a tumor of this type situated in any other part of the body nearly every surgeon would select excision as a first choice and the mere fact that the tumor is in the lung does not alter this principle. This I have repeatedly stated and, indeed, made it the subject of my presidential address before this association in Chicago in 1924.[2]

There are a number of conditions, however, which might and should contraindicate a pulmonary operation dangerous to life. There are first, the presence of another disease as serious or even more serious than the one which we are treating, such, for example, as a progressive and probably fatal circulatory disorder or, generally speaking, the existence of another malignant growth. Yet even here there are exceptions; for example, the one which we felt should be excluded when the urinary system of our patient was examined by x-ray; namely, hypernephroma.

for here metastasis may be solitary and its extirpation may be fully justifiable with the removal of the parent growth at the same time or at a later period.[3]

This, of course, is the exact opposite of the manner in which operations for the more usual malignant tumors are undertaken. We will not go further into this general question but will now discuss the propriety of operating for tumor of the lung in the presence of tuberculosis of that organ.

We must again compare the danger to life of the two coinciding ailments. It is obvious that malignant tumors of the lung in the presence of active pulmonary tuberculosis should be treated according to the rate of progression of each disease. In the presence of chronic phthisis with an outlook for prolonged life it would seem quite natural to disregard the tuberculous infection.

In the case of Miss K., the tuberculosis was supposed to be dormant and, in fact, its presence was apparently not the cause of death nor even of the fatal bronchopneumonia. The absence of postoperative cyanosis and the slow respiratory rate gave every reason to believe that the remaining area of functioning lung would be ample to sustain life, provided no accident, such as the bronchopneumonia, should occur. The open tuberculosis was found in the left (sarcomatous) lung, not in the remaining one. Had I known, however, that there was a tuberculous cavity in the right lung even though a closed one, I would probably have refrained from radical operation.* Even the modern method of examining the gastric contents for bacilli would probably not have been positive, though in my next case of this kind I would not omit this form of examination in addition to the very reliable concentration method.

It must be remembered that even though a carcinoma of the lung may be inoperable, much may be done to relieve suffering and to prolong life. I hope to write a paper describing some of these methods at some future time. This, however, has little to do with the subject presented today.

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*In a letter discussing this case Dr. Amberson writes: "I do not agree with you in your statement that a radical operation would have been contraindicated if you had known there was a tuberculous cavity in the right lung. The important thing is whether this is well encapsulated or whether it is recent and ready to rupture into a bronchus. It is quite probable that this cavity was a remnant of the original one. So long as it remained closed for so many years and was not shedding tubercle bacilli into the bronchi, I believe that the chance we took was justified in view of an otherwise hopeless situation. I am not at all satisfied from the pathological report that this patient was giving out tubercle bacilli. It seems to me that all these areas, most of which we *knew* were present, were in reality closed foci. I should expect of course that some of them might have been rendered more vascular and softer by the bronchopneumonia, even though this was only of short duration."

He also says, in estimating the criteria of healing or arrest, that, "a—Absence of tubercle bacilli from the sputum on examination by concentration method. (This is a pretty dependable thing if the patient is trained not to swallow the sputum and, in a case like Miss K.'s, I do not see that there would have been any particular advantage in an examination of gastric contents.)

"b—X-ray evidence of stationary or retrogressive lesion without demonstrable cavity assuming that serial x-rays are made from time to time.

"c—Ability to live practically a normal life without symptoms over a period of years."

CONCLUSIONS

1. The mere presence of pulmonary phthisis is not of itself a contraindication to operation, radical or palliative, in malignant diseases of the lung.

2. Active and progressive tuberculosis should contraindicate operation of this type except to tide over an emergency.

3. The character of the operation should depend upon the size, location, and rapidity of progress of the malignant disease.

4. The ability of the patient to withstand a major surgical procedure and the extent of the operation and its contemplated value must be carefully estimated by the surgeon. The vital capacity, blood pressure, nutrition, and the presence or absence of cough and fever, and the presence of other diseases must be taken into consideration.

5. Preoperative bronchoscopy is essential.

6. The presence of another neoplasm need not be considered a contraindication to operations upon the lungs unless it directly threatens life.

7. The choice of the anesthetic and its method of application is of importance. Unless there is profuse expectoration, avertin in a basal dose is invaluable.

8. The treatment of tumors of the lung should not be different from that of tumors in other vital parts of the body. A properly performed exploratory thoracotomy is less shocking than a similar operation upon the abdomen.

9. To wait until suppuration occurs will probably be fatal.

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DISCUSSION ON "BRONCHOGENIC CARCINOMA," BY DR. CLERF, AND "PNEUMONECTOMY FOR SARCOMA OF LUNG IN A TUBERCULOUS PATIENT," BY DR. LILIENTHAL

DR. EVARTS A. GRAHAM (ST. LOUIS).—I was much interested in this case of Dr. Lilienthal because he operated upon his case just a short time before I performed a complete pneumectomy. In my case, however, fortunately the result was successful. I do not call it pneumonectomy, as Dr. Lilienthal does, because I have the support of the Oxford dictionary and various other dictionaries to call it pneumectomy instead of pneumonectomy.

My patient was 47 years of age, a physician, who for six months had had symptoms of an atypical form of inflammation of the lung, involving chiefly the left upper lobe. An attempt had been made elsewhere to aspirate what was regarded as an interlobar empyema. An accidental pneumothorax occurred. The patient's

attending physicians maintained artificial pneumothorax, but the symptoms later began to get worse. At the time when Dr. Singer and I first saw him he had a complete atelectasis of the left upper lobe with pneumothorax around it. It was quite apparent that anybody with an atelectasis of that sort must have a bronchial obstruction, and in a man of that age with an insidious onset, there seemed to be more than a 95 per cent chance that his bronchial obstruction was due to a tumor which was probably a carcinoma.

The patient had a very phlegmatic disposition and when he insisted on knowing the diagnosis we told him that we suspected a carcinoma of the bronchus but that the suspicion ought to be confirmed by bronchoscopic examination, which was done by Dr. Arbuckle. A biopsy was made on some of the removed tissue and a diagnosis of squamous cell carcinoma was made. He then wanted to know what to do about it. I told him that radiotherapy had never cured an authentic case; the only thing that I thought would offer him any chance was operative removal.

It was intended to perform a lobectomy of the upper lobe. But after getting into the pleural cavity an opportunity to examine the lung properly showed several things. In the first place, there were numerous nodules in the upper part of the lung which I thought were probably inflammatory. There were also nodules in the upper part of the lower lobe, the nature of which was uncertain. Moreover the interlobar tissue was not complete. In other words, it would have been necessary in doing a lobectomy to cut through inflammatory tissue in the upper part of the lower lobe. Finally, on palpation of the bronchus it was determined that it would not be possible to get above the carcinoma of the upper lobe bronchus without sacrificing also the lower lobe bronchus. Accordingly, it was decided to perform a complete pneumectomy. The upper lobe was adherent by several dense vascular adhesions and the lower lobe was completely adherent to the chest wall. Separation, however, occurred without undue difficulty. The lung was well mobilized. The excision of the lung was made as close to the bifurcation of the trachea as possible. In this manner I was able to get above the tumor by a distance of about 1.5 cm. I also removed several enlarged mediastinal glands which, however, on microscopic examination revealed no spread of the carcinoma.

After the removal of the lung it was thoroughly examined by microscopic studies made on tissue taken from various parts of it. There was a squamous cell carcinoma involving the bronchus of the upper lobe for a distance of about 1.5 cm. which had extended right up to the bifurcation of the main bronchus, so that the bronchus to the lower lobe could not be saved. It was especially interesting, however, that the tumor in no place had passed through the cartilage and that no evidence of a spread to the pulmonary parenchyma was noted. The nodules in the lung which had been palpated at operation were all inflammatory, small abscesses.

Seven ribs (the third to the ninth inclusive) were removed throughout almost their entire length in order to obliterate the pleural cavity as much as possible. The first and second ribs were not removed at that time because it seemed safer to postpone their removal until a later time. The hilus was doubly ligated *en masse* with transfixion ligatures of chromic catgut, after separately ligating the pulmonary artery, and the mucosa of the bronchial stump was cauterized thoroughly with an electric cautery. The anesthesia used was intratracheal nitrous oxide and oxygen.

The immediate postoperative course was almost uneventful. Drainage of the pleural cavity through an air-tight catheter had been instituted, and for the first four or five days about 500 c.c. of serum were drained off. On about the tenth day an empyema developed in the small remaining portion of the pleural cavity at the apex where the presence of the remaining first and second ribs prevented a collapse of the pleural space. There was also a small communication between this empyema cavity and the bronchial stump. The apical cavity was drained anteriorly in order

not to infect the back, and about a week later the first and second ribs were removed through a posterior incision. Prompt obliteration of the cavity followed this operation, and the empyema rapidly became healed. At the time of revising this discussion (June 10) the wounds are healed, the patient is walking around, and he plans to return to his home in one week. His vital capacity is 1500 c.c. and there is moderate dyspnea after walking up and down stairs.

Apparently this is the first case in which a whole lung has been removed successfully for carcinoma. Not only is the immediate recovery from the operation gratifying, but it is also particularly encouraging that no spread of the carcinoma to the regional lymph glands or to the parenchyma of the lung could be demonstrated. There seems to be some basis for hope, therefore, that all of the carcinoma has been eradicated. Moreover, it is possible that future experience will determine that better results will follow the removal of an entire lung for carcinoma than merely a single lobe, by analogy with operative results on other organs.

DR. CUTLER.—How did you treat the bronchial stump?

DR. GRAHAM.—I was afraid it would take up too much time to go into the treatment of the bronchial stump in detail. Of course, we had no lung tissue with which to cover it because of the removal of the whole lung and also because I desired to take the bronchus out as close to the trachea as I could. So there was nothing left to do except to ligate it. I ligated the bronchus twice with No. 2 chromic catgut, ligating the pulmonary artery separately. Before pulling the sutures tight on the bronchus, however, I cauterized the mucous membrane as high up as I could reach with an electrocautery, destroying, I think, the entire mucous membrane before pulling the sutures tight around the bronchial stump. I also implanted five gold radon seeds into the peribronchial tissues.

DR. CORYLLOS.—What is the thoracoplasty you have done in the section of the seven ribs?

DR. GRAHAM.—I did the thoracoplasty at the same time. I took out seven ribs at the time of the operation. That, of course, was an extra procedure, and I can see its dangers, but as a matter of fact, this man left the operating table with the same pulse and blood pressure that he had when he arrived.

One may well ask, perhaps, why not the thoracoplasty first and the pneumectomy later, in two stages? I can see an objection to that order of events. If one does the thoracoplasty first he will be collapsing the chest wall down upon the lung. There will therefore be some danger that later when the pneumectomy is performed, the enormous pleural space will not be completely obliterated. If it is not completely obliterated there will almost certainly be an empyema communicating with an opening in the bronchial stump. It seems to me that the closure of the bronchial stump in a case of complete pneumectomy cannot be satisfactory unless the soft tissues of the chest wall are allowed to fall in upon it so that the parietal pleura will become solidly healed to it.

DR. CARL A. HEDBLOM (CHICAGO).—I should like to express my great admiration over the wonderful result Dr. Graham has just reported.

May I also stress the importance of early diagnosis and of not placing undue reliance on negative findings. Three weeks ago I did an exploratory thoracotomy on a woman about forty years of age who had developed hemoptyses seven months before. The outstanding positive finding was a clouding of the left base. No bacilli were found in the sputum. Two bronchoscopic examinations were performed the last one just before thoracotomy with excision of tissue which showed no evidence of malignancy. At the exploratory thoracotomy extensive carcinoma was found in-

volving both lobes, the diaphragm and pericardium and the glands at the hilus were greatly enlarged. Had the operation been performed in the first week of illness an excision of the lower lobe would have given her a chance for a cure.

Possibly a ligation lobectomy will prove the operation of choice in some of these cases.

POL. N. CORYLLOS (NEW YORK).—Illustrating on blackboard. First a point of etymology. I agree with Dr. Lilienthal that the correct Greek term for excision of the lung is "pneumonectomy" and not "pneumectomy" as proposed by Dr. Graham. The lung in Greek is *πνεύμων-ονος*, whereas *πνεῦμα-ατος* means air or spirit. The second part of the term, "*εκτομὴ*" (excision) indicates the action exerted upon the lung, therefore the latter must be placed in the genitive (*πνεύμονος*) the radical of which is *πνεύμον-* so that the composed word will be "pneumonectomy." "Pneumectomy" on the contrary means "resection of air," and if correctly constructed should be pneumatectomy. It is according to the same rules that are constructed the terms *gastrectomy, ureterostomy, salpingectomy, oto-rhino-laryngology*, etc. I hope that this literary digression will help to settle that question.

I want to congratulate Dr. Lilienthal and Dr. Graham for their attempts to resect the entire lung for cancer. The comparative study of these two cases is of great interest. In fact I am disposed to believe that the success of Dr. Graham is greatly due to the resection of the ribs by which he terminated his operation, which was not done by Dr. Lilienthal. The collapse of the chest wall in the case of Dr. Graham brought about the obliteration of the pleural cavity, left empty after the resection of the lung, and thus reestablished to a considerable extent normalcy in the mechanics of the other lung. Whatever may be the pressure in the empty pleural cavity after pneumonectomy, equal, inferior or superior to one atmosphere it will hamper the normal function of the other lung. This state of affairs is certainly aggravated by the development of fluid in the pleural cavity. Postoperative pneumonia follows bronchial stasis and this is favored by disturbance in the normal movements of the lung. It seems also very probable that the intratracheal anesthesia used by Dr. Graham has a part in his success. As I have already pointed out, I am convinced that this mode of anesthesia by maintaining patent the respiratory ways, preventing rhinopharyngeal secretions from flowing into the lower respiratory tract and by allowing suction of the bronchial exudate before, during and after operation, helps to prevent anoxemia, infection of the bronchi and bronchial stasis.

I wonder however whether it would be preferable to resect the ribs in a first stage and proceed to the pneumonectomy 8 to 10 days later. I am afraid that extensive thoracoplasty and pneumonectomy performed at the same stage is too great a burden for the patient.

DR. DEAN B. COLE.—I wish to ask Dr. Lilienthal and Dr. Graham what is the life expectancy if operation is successful? What is the prognosis for the patient Dr. Graham reported and what are the probabilities of recurrence?

We see many patients with lung cancer and find it difficult to advise them relative to treatment. Our results from x-ray therapy and implantation of radium are disappointing. On the other hand, we see an occasional patient with proved lung cancer who lives in comparative comfort for years without treatment.

DR. HOWARD LILIENTHAL (NEW YORK CITY).—I had hoped to bring out discussion on the matter of operating upon lungs that were tuberculous. I did not present my case merely as one of cancer of the lung. I reported it as a cancer of the lung in a *tuberculous individual*, and the question that I wanted to have

discussed and hoped would be discussed was, what are our rights in operating in a case in which a tuberculous patient has a tumor of the lung?

We have heard from Dr. Kinsella that other parts of the body of tuberculous patients can be operated upon safely, but his cases were in individuals who had been cured of tuberculosis by surgery.

Now here is a patient who was well as far as we knew—pronounced well by authority—and the question that I wanted to bring up was, were we right or wrong in trying to get rid of the cancer of the lung, assuming that it was as we believed it to be, a primary neoplasma.

Referring to the remarks of Dr. Cole regarding prognosis, I should think that the removal of all of the cancer especially when the lung is also resected should be at least as good as that when breast cancer has been removed by mastectomy.

Now about patients that get well in spite of the diagnosis of cancer of the bronchus and without extirpation. I have a female patient whom I did not operate upon because she had what was pronounced a carcinoma too near the trachea, I thought, to promise success. She was in her twenties, and that was five years ago. She was treated with radium to the growth and radium pack without. I then suggested lower lobectomy because of suppuration. She is alive and has been working ever since, and now I find on submitting the sections to Dr. Paul Klemperer of the Laboratory of Mt. Sinai Hospital, who had not seen the specimens in the past, that this was a strange form of bronchial tumor which used to be regarded as carcinoma but which was not carcinoma.

CHANGES IN THE HEART AND PERICARDIUM BROUGHT ABOUT BY COMPRESSION OF THE LEGS AND ABDOMEN*†

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AND
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ALMOST all cases of trauma to the heart and pericardium are the result either of a penetrating wound or of a compression injury to the chest. It is known that a blow or a kick upon the precordial portion of the thoracic wall may bring about rupture of the heart even though the ribs or sternum is not fractured. That the heart and pericardium can be ruptured by the sudden application of a compression force limited to the abdomen and lower extremities seems to be impossible on a priori considerations. Nevertheless, the literature contains a few examples of cardiac trauma brought about by indirect forces.

Kellert[2] reported the case of an Italian, forty-four years old, to whom an accident occurred while he was engaged in digging in a sand bank. His fellow workmen heard him cry out and when they turned about, they found that the sand bank had caved in and that the man was surrounded up to his waist by a large quantity of sand. He was unconscious, but apparently alive. When dug out, he was dead. Extensive damage to the heart and aorta was found. A laceration 5 cm. in length extended through the interventricular septum and opened both ventricular cavities. The papillary muscles in both ventricles were torn. The endocardium of the right auricle was lacerated. The aorta at its origin was separated through one-half the circumference as though it had been incised. The pericardium was ruptured. Just beneath the right auricle were two minute openings in the fat and myocardium. In the left ventricular wall beneath the auricular appendage was a transverse laceration in the muscle 2.5 cm. long. Microscopically, there was a slight amount of fatty infiltration of the right ventricular wall and vacuolization of the muscle fibers with increased amount of pigment about the nuclei, which varied greatly in size. An occasional area of fibrosis and a rare focal accumulation of lympho-

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cytes, endothelial cells and polymorphonuclear leucocytes were seen. According to Kellert,

"There are two possibilities in explanation of the lesion in the present instance. First, that the weight of the falling earth, which being soft and adaptable, caused equal pressure over the chest resulting in great compression and bursting of the heart, very much as though a rubber bag distended with fluid were compressed at its middle. To obtain such an effect without fracture of the sternum or ribs seems very unlikely. [Furthermore Kellert stated that the patient was engulfed only up to the waist.] The second and more probable explanation is that of hydraulic pressure. The large quantity of sandy soil exerted such great pressure over the lower half of the body as to drive most of the blood out of the vessels. This produced sudden over-distention of the heart, which was probably dilated as a result of physical exertion, with consequent rupture at several points. That the force was a great one is indicated by the ruptured pericardium and hemorrhages in the lung. The pathologic changes in the heart, though not very marked, undoubtedly contributed to the severity of the injury."

Saphir[3] reported the case of a boy, four years old, who was run over by a motor truck. The thoracic wall showed no evidence of injury. The pericardial cavity contained about 300 c.c. of blood. The left ventricular wall was ruptured for a distance of 6.5 cm. The myocardium showed no evidence of disease. The pelvis was fractured. Saphir stated: "While blows to the chest cannot be excluded, we saw no evidence of injury to the chest wall. Therefore the most probable explanation is that the rupture of the heart is dependent on the trauma of which the greatest force was exerted in the region of the right pelvis."

Stephens[5] briefly recorded an example of rupture of the heart as follows: "A well-built lad, aged sixteen, was standing near a crane from which was suspended a heavy steel tube. As the tube was lowered it pressed against his chest and abdomen; he then stepped back a few paces and fell down, dead. Postmortem examination showed the intrapericardial pressure was atmospheric, and on opening the sac it was found to be full of blood. In the right auricle was a tear one inch long. Was this due to the back pressure caused by the tube pressing on his abdomen?"

EXPERIMENTS

Whether the heart can be injured by such indirect forces is a moot question and cannot be answered by any experimental work that we have been able to find. We, therefore, carried out experiments in which pressure was applied to the legs and abdomen and the compression effect upon the heart studied.

Fig. 1.—*A*. Control roentgenogram. *B*. Roentgenogram of the heart immediately after application of 12 cm. pressure to hind legs and abdomen. *C*. Roentgenogram of the heart after pressure had been applied for two minutes. The heart dilated as soon as pressure was applied and then decreased slightly in size. The dilatation was chiefly on the right side of the heart.

METHOD

Dogs were anesthetized with ether. The skin was shaved just above the umbilicus completely around the body over an area about 6 cm. in width. A transverse incision about 8 cm. in length was made midway between the umbilicus and sternum and carried through the skin and platysma. The skin was then undercut around the circumference of the body, and a metal band about 4 cm. in width was placed around the body and the skin was sutured over the band. The hind legs and abdomen were inserted into a rubber bag which was encased in an outer bag of canvas. The rubber bag was then hermetically closed upon the metal band by applying a tightly wound rubber tourniquet. The metal band placed around the body and under the skin supported the tourniquet which had to be wrapped tightly, and the band prevented the tourniquet from interfering with the circulation through the aorta and vena cava. Heavy pressure tubing connected the rubber bag to the air pressure main. When the air pressure was turned on, the bag became inflated and exerted a compression effect upon the hind legs and the abdomen. It required about fifteen seconds for the pressure in the bag to rise to 120 mm. Hg. Pressures varying from 120 to 600 mm. Hg were used in the experiments.

OBSERVATIONS

The Effect Upon the Size of the Normal Heart Brought About by Compression of Legs and Abdomen.—The size of the heart was determined by roentgenograms which were standardized by having the target and plate at fixed distances from the heart. The dog having been anesthetized was maintained in a true anteroposterior position throughout the experiment. The exposure was one-twentieth of a second and was made at the end of expiration. With these precautions slight variations in the size of the cardiac shadow occurred, but in the controls these did not measure more than 2 mm. in the transverse diameter of the heart. This variation undoubtedly was accounted for by the diastolic systolic excursion of the heart.

Twenty observations were made on 9 dogs in which the size of the heart was determined immediately after pressure had been applied to the legs and abdomen, and again after the pressure had been on for two minutes. In every experiment the cardiac shadow underwent an enlargement (Fig. 1). The average increase in the transverse diameter of the cardiac shadow was 2.3 mm.; the greatest increase in any of the experiments was 10 mm. After the heart underwent this initial increase in size, the cardiac shadow decreased slightly in size as the pressure continued to be applied. This decrease in size came about as the circulation accommodated itself to the changed conditions. The enlargement of the cardiac shadow brought about by a compression force of 120 mm. Hg was about the same as that produced by 170 or

Fig. 2.—Roentgenograms taken after the pericardium had been incised. *A*, Control before application of pressure. *B*, Immediately after application of pressure of 12 cm. Hg. to legs and abdomen. *C*, After the pressure had been applied for two minutes. The dilatation of the heart in response to this pressure was greater in the experiments in which the pericardium had been incised than in those in which the pericardium was intact. The pericardium may restrict acute dilatation of the heart.

200 mm. Hg. This enlargement of the cardiac shadow was about the same as that produced by the administration of 1000 c.c. of normal salt solution intravenously.

The contour of the cardiac shadow also changed after the legs and abdomen were compressed. The heart became more globular, and particularly the right ventricle became more prominent.

The Effect of Compression of Legs and Abdomen Upon the Size of the Heart After the Pericardium Had Been Removed; Also the Effect After Plethora Had Been Established.—In these experiments the chest was opened and the pericardium was incised from the apex to the base of the heart. The opening in the chest was then closed and the air was removed from the pleural cavity. Roentgenograms of the heart were taken before and after the application of pressure to the legs and abdomen.

It was found that the heart responded to compression of the legs and abdomen by enlarging on an average of 9 mm. in its transverse diameter (Fig. 2). The greatest dilatation of the heart was 19 mm. It was also found that the heart did not return to its normal size after pressure had been applied for several minutes with the same regularity as did the heart with the pericardium intact.

To exclude the effect of pneumothorax upon the circulation, three experiments were carried out in which the pericardium had been removed by operation two weeks before these determinations were carried out. The effect of compression of the legs and abdomen in these experiments was similar to the results given above.

A condition of plethora was then established by the administration of from 500 to 2000 c.c. of salt solution intravenously. One liter of this solution given to a large dog caused a dilatation of the heart measuring about 5 or 6 mm. in the transverse diameter. After plethora had been established, compression of the legs and abdomen resulted in a marked dilatation of the heart (Fig. 3); this enlargement averaged 15 mm. The greatest dilatation was 20 mm. However, rupture of the heart did not occur in any of these experiments.

The Effect of Compression of the Legs and Abdomen Upon Arterial, Right Auricular, Intrapericardial, and Intrapleural Pressures.—In this group of experiments the pericardium had been sutured to the thoracic wall over an area about 4 cm. in diameter so that, after healing of the wound had taken place, the pericardial cavity could be entered without opening the pleural cavity. In this way pneumothorax was avoided when the pressure determinations were made. The arterial pressure was obtained from one of the carotid arteries. The right auricular pressure was obtained through a metal cannula inserted into the auricle through a jugular vein. The intrapleural pressure was obtained through a metal catheter inserted into the anterior part of the right chest. The intrapericardial pressure was obtained through

Fig. 3.—Plethora had been established by the administration of 500 c.c. of salt solution intravenously. *A.* Control. *B.* Immediately after application of 12 cm. Hg pressure. *C.* After pressure had been applied for two minutes. A marked dilatation occurred after the application of pressure in those experiments in which plethora had been established.

a metal catheter inserted into the pericardial cavity through the scar of the previous operation.

When a pressure of 120 to 180 mm. of mercury was applied to the hind legs and abdomen, the following observations were made (Fig. 4): 1. The arterial pressure in five experiments rose from a few

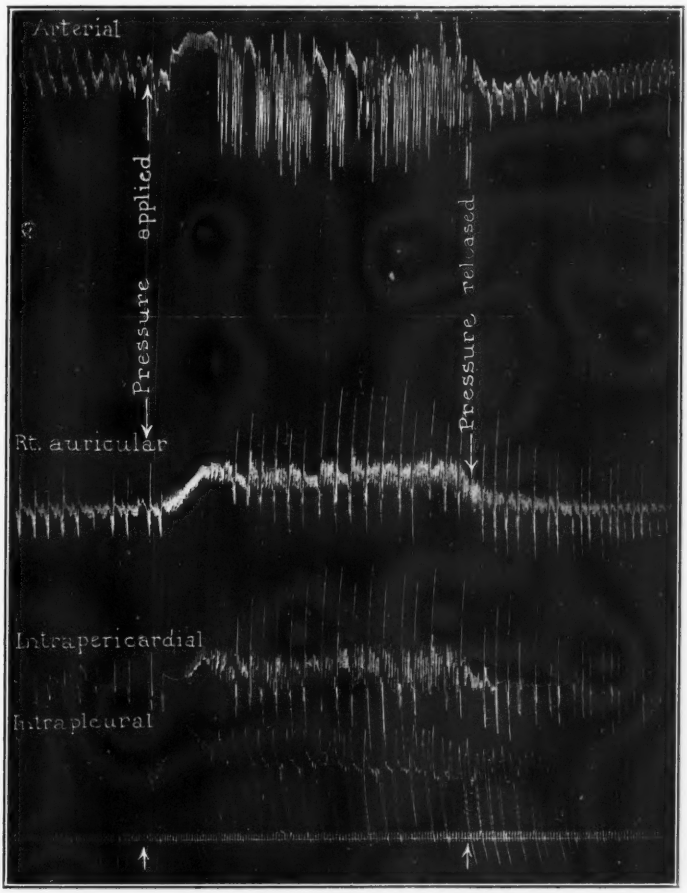

Fig. 4.—Graph showing arterial, right auricular, intrapericardial, and intrapleural pressure changes brought about by a compression force of 180 mm. Hg applied to the legs and lower abdomen.

millimeters of mercury to 34 mm. mercury as soon as pressure was applied. From this initial rise the pressure fell, and sometimes it fell below the normal level. When the compression was released the pressure temporarily fell below the normal level. 2. The right auricular pressure rose in each experiment and remained elevated as long as the pressure was being applied. The average rise in auricular pres-

sure measured 40 to 80 mm. of normal salt solution. Throughout the period of compression the excursion of the tambour was definitely increased, indicating greater swings in the auricular pressure. 3. The intrapericardial pressure increased in each experiment from 40 to 70 mm. of normal salt solution. The swings of pressure coincident with the heartbeat were exaggerated. 4. The intrapleural pressure rose several centimeters of water and the respiratory excursion was usually increased.

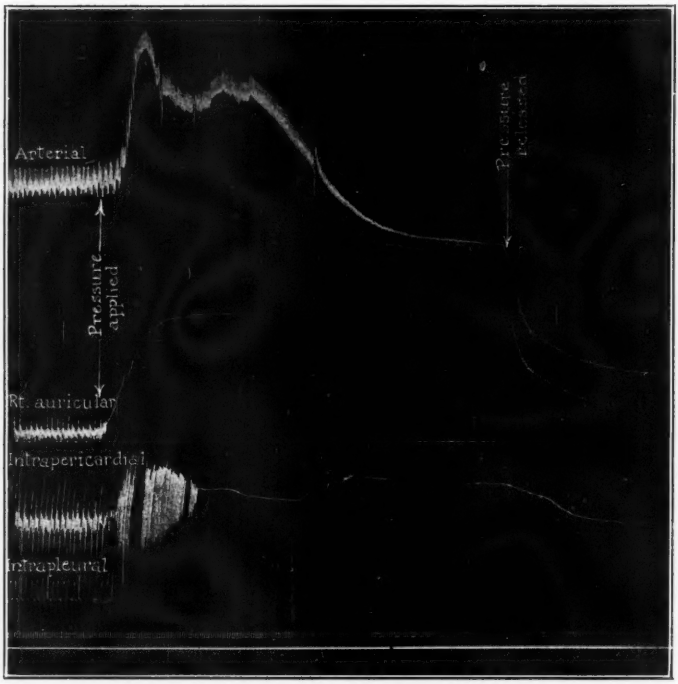

Fig. 5.—Graph showing arterial, right auricular, intrapericardial, and intrapleural pressures in an experiment in which plethora had been produced by the administration of 2000 c.c. of normal salt solution. A compression force of 180 mm. Hg was applied to the legs and abdomen. It brought about failure of the cardiorespiratory mechanism.

In the experiments in which plethora was produced by the administration of 1 or 2 liters of normal salt solution, the alteration in the pressures obtained by compression of legs and abdomen referred to above were greatly exaggerated. Compression brought about an immediate rise of arterial pressure measuring as much as 110 mm. Hg. The administration of 1000 c.c. of normal salt solution intravenously caused a transient average rise of 40 mm. in the right auricular pressure, while compression after plethora had been produced caused an immediate rise in right auricular pressure measuring as much as 300

mm. of normal salt solution. In one experiment in which plethora had been produced, the application of pressure to the legs and abdomen produced a sharp rise in arterial, right auricular, and intrapericardial pressures; respiration ceased as the pressure was applied and the circulation gradually failed (Fig. 5). This was the only example of cardiac failure obtained in these experiments.

Arterial and Venous Pressure Determinations in the Lower Abdomen During the Period of Compression.—The blood flow in that part of the body that was being compressed was determined by passing a non-collapsible ureteral catheter through a jugular vein and through a carotid artery into the lower abdomen. When these catheters were connected to manometers, it was found that the arterial and venous pressures were increased with each increment of pressure in the rubber bag, but the pulsation in the artery ceased when the pressure in the bag rose above the level of arterial pressure. The cause of this rise in pressure was due to the wall of the blood vessel being forced into the mouth of the catheters. When the catheters were connected to a syringe, blood could not be drawn from either artery or vein after compression was applied, indicating that the arteries and veins were collapsed and did not contain blood.

NECROPSY FINDINGS

In no experiment did the clinical manifestations of traumatic asphyxia develop. Ether narcosis was used. In those experiments in which plethora was not established, the necropsy findings were of little significance. An occasional petechial hemorrhage was found in the epicardium and in the fat at the base of the aorta. In one experiment gas passed freely through the gastrointestinal tract and was forced out through the mouth in large quantities. In this experiment the large bowel had longitudinal tears in the mucosa and serosa, and the abdominal cavity contained some free blood. In those experiments in which plethora had been established small hemorrhages usually were found in the epicardium, in the myocardium of both ventricles, most frequently along the left coronary artery, in the epicardial fat at the base of the aorta, and in the wall of each auricle. In one case marked endocardial hemorrhages were produced. In two of the experiments complicated by plethora the urine was bloody and the mucosa of the bladder was hemorrhagic; in one experiment definite hemorrhagic areas in the lung were present, and in another, hemorrhagic areas were found in the stomach, intestines, and mesentery. The intestines frequently appeared pale, and in one experiment in which the animal was killed while compression was applied and the aorta and vena cava were clamped above the diaphragm, the intestines were extremely pale and almost completely bloodless. In no experiment was the diaphragm ruptured.

DISCUSSION AND SUMMARY

A compression force suddenly applied to the legs and abdomen manifests itself primarily by occluding the aorta below the diaphragm and rapidly forcing the blood out of the veins of the legs and abdomen into the reservoirs above the diaphragm. The collapse of both arteries and veins in the compressed parts was shown by experiment. This mechanism, therefore, is equivalent to removing part of the vascular bed from the circulation and throwing the quota of blood that normally goes to these parts into the rest of the circulation. The severity of this burden on the circulation depends upon the proportionate quantity of blood displaced and the ability of the heart and vascular bed to receive it. The anesthetized dog responds to this alteration in the circulation by an immediate but temporary dilatation of the heart, especially of the right ventricle, a rise in the right auricular pressure, and a temporary rise of arterial pressure. In our experiments the heartbeat became much more forceful than normal and this was shown by the increased oscillations in the intrapericardial, intra-auricular and arterial pressures. Two factors probably brought about these changes, namely, removal of part of the vascular bed and an engorgement of the right ventricle with blood. The peripheral bed of that part of the circulation which still carried blood soon adapted itself by a compensatory dilatation, and the heart became somewhat smaller. This adaptation, however, was not complete because the right auricular pressure remained elevated and the heartbeat remained forceful as long as the pressure was applied. The response of the circulation to compression of the legs and abdomen was not unlike the response obtained by increasing the circulating blood volume. That the pericardium supports the heart as it dilates in response to this sudden alteration in the circulation was shown by experiment. If the circulating blood volume had already been increased by the administration of salt solution, then the response to compression was greatly exaggerated. After plethora was produced, the heart underwent an acute dilatation of extreme degree upon compression of the legs and abdomen. In one experiment the heart failed.

The only evidence of trauma found at necropsy in the experiments in which the legs and abdomen were compressed was the presence of a few small hemorrhages in the epicardium and myocardium, hemorrhage in the lung, in the bladder, and in the mesentery of the intestine. This evidence of trauma was not always found. Although the heart was markedly dilated at death, it was never ruptured. The diaphragm was elevated the distance of two ribs by the compression, but it was not ruptured.

We should like to answer the questions as to whether such forces as a compression of the legs and abdomen can in any circumstances

bring about rupture of the heart. In the experiments we obtained a marked dilatation of the heart and a marked elevation of the right auricular pressure. In one experiment compression of the legs and abdomen brought about circulatory failure, but actual rupture did not occur. If we may express an opinion, we would say that under certain circumstances the transfer of back pressure in the aorta and vena cava may be sufficient to rupture either auricle or ventricle. The circumstances that seem to determine this form of cardiac rupture are not alone the degree and suddenness of the compression but also certain characteristics of the circulation and cardiac muscle. These characteristics are the rapidity of the circulation at the time the pressure is applied and the degree of elasticity and friability of the myocardium. It is our opinion, based upon experiments in dogs, that the degree of friability of living cardiac muscle is a variable property and it may be assumed that the elasticity and friability of the human heart are also variable factors. If the circulation is overburdened when the pressure is applied to the legs and abdomen, the heart may be brought to a standstill. If the myocardium is abnormally friable, compression of the legs and abdomen presumably may bring about rupture of the heart.

This indirect type of force applied to the heart is not unlike the type of injury that produces traumatic asphyxia. Heuer's résumé[1] on this subject does not refer to rupture of the heart as having occurred in any of the cases of traumatic asphyxia. However, in several cases of traumatic asphyxia hemorrhages were found in the pleura and in the epicardium, and these findings are similar to the results obtained in the experiments recorded here. Our finding of marked engorgement of the right side of the heart in these experiments is in agreement with the observations made by Shimonek[4] and lend support to his ideas of therapy for traumatic asphyxia. His patient was unconscious and had stopped breathing. "The heart was beating against the chest wall with great vigor pointing to the great obstruction against which it was laboring. It instantly occurred to me that to save his life the right heart would have to be relieved forthwith of the immense burden, then to institute artificial respiration if it did not promptly respond to the emptying of the venous system." After a vein was opened ". . . the blood flowed in a large black stream and almost immediately the color of the patient's face began to lose its blackness, the protrusion of the eyes soon lessened. . . . There is no doubt in my mind that the prompt and copious venesection saved his life."

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DISCUSSION

DR. GEORGE HEUER (NEW YORK).—The paper to which Dr. Beck referred was one on Traumatic Asphyxia which I published in 1923. I assembled 127 cases of this condition and studied their clinical and necropsy records. Although the compressing force was exerted upon the chest or abdomen or both, the number of cases in which there were serious intrathoracic injuries was rather small.

In the cases which recovered, hemoptysis occurred in 16, hemothorax in 3. In the necropsy records I found hemothorax in 6 or more cases, rupture of the lung in a few cases and occasionally subpleural and subpericardial hemorrhages. In no case did I find rupture of the pericardium or heart.

The subject is interesting from a clinical standpoint. During the War we saw quite a number of cases of penetrating wounds of the thorax in which there seemed to be evidence either of injury to the heart or injury to the pericardium. Among them were instances of hemopericardium, pneumopericardium and hemopneumopericardium. Fluoroscopic examination in some of these showed a foreign body which moved with the cardiac movements; and the question always arose whether the foreign body was in the heart, in the pericardium or just outside the pericardium and moved by the cardiac impulse.

Under conservative treatment the majority of the cases recovered. In a few instances in which we explored the pericardium we found hemothorax and hemopericardium but in no instance in my experience did we find a penetrating wound of the heart.

DR. EDWARD D. CHURCHILL (BOSTON).—I think there may be another clinical application of these very beautiful experiments of Dr. Beck's. I refer to the cases of Pick's disease, in which we remove the pericardium and at times the thickened scar on the surface of the heart, leaving a very thin walled ventricle which has suffered an unknown amount of damage from the adherent scar. Such a ventricle may well be damaged or ruptured by a marked increase in the return of blood to the heart. In just this particular I have differed with Dr. Beck in the conclusions drawn from his previous experimental work, because he feels that the danger may lie in a reduced cardiac output from pneumocardia tamponade.

Personally, I have preferred to have the cardiac output cut down very markedly in the immediate postoperative period in such a situation, because I do not want to subject the heart to even its normal cardiac output with the right ventricle looking the way it does sometimes after the scar has been dissected away from it. I would prefer to operate on these patients in a sitting position, which somewhat reduces the return of blood to the heart. I have deliberately left a partial pneumothorax for the same purpose.

Transfusion is to be avoided, even in the face of a low blood pressure, and at the very slightest sign of venous distention I should not hesitate at all further to reduce cardiac output by a liberal venous section until the weakened myocardium has a chance partially to regain its support from the scarred tissue which forms after the operation, or until it has had a chance to reëstablish its normal tonus. So far as I recall, there has been no account of this accident occurring postoperatively in decortication of the heart although it may have been unrecognized in certain of the fatal cases.

THE REMOVAL OF A NEEDLE FROM THE HEART WITH ELECTROCARDIOGRAPH RECORDS BEFORE, DURING, AND AFTER OPERATION*

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THE removal of a needle from the heart is still a sufficiently rare event in itself to warrant interest. In the case here reported there is the additional interest of a fairly accurate history of over six months from the time of injury to operation with the gradual development of symptoms; an unusual situation of the needle transfixing the heart from right auricle to left ventricle; the opportunity to record electrocardiographic readings before and during operation, and after removal; together with certain technical difficulties which led to the adoption of a method of removal which might prove of value on other occasions.

The points of importance in the history are as follows:

Five months before her admission to the Royal Victoria Hospital, a perfectly healthy young woman tripped and fell forward on her face. In falling a darning needle which she had stuck into the front of her dress, was driven into her left chest, about the level of the fifth rib, near the sternal border. The needle disappeared completely from sight. As she rather naïvely stated, since she had no money and it did not hurt much, she did nothing about it at the time. During the following weeks she became increasingly troubled by a pain in the chest. It was described as sharp and severe. It gradually became more severe and was present constantly day and night. At times it radiated across the front of the chest on the left side but never down either arm or to the back.

The pain was increased on exertion, so that for the last two months she had been unable to work. One month previous to admission, the pain became so severe on exertion, that she was completely incapacitated. About this time she began to have some difficulty in breathing, which added greatly to her distress.

She then consulted a physician, and a radiograph disclosed a needle-like shadow within the cardiac area. She was referred to the Royal Victoria Hospital by Dr. A. V. Traynor of Kitchener, Ontario.

She had always been strong and healthy and worked hard. She was well developed and examination revealed nothing abnormal except in the cardiovascular system. The pulse rate was 80 and regular in rhythm. There was no evidence of arterial thickening. The blood pressure was 130 mm. of mercury systolic and 94 diastolic. There was no evidence of increased intravenous pressure. The heart was slightly enlarged to the left. The sounds were of normal intensity. There were no murmurs present. There was no precardial friction rub nor evidence of increase of fluid in the pericardium. The lungs were normal. The liver was not palpable. There was no peripheral edema. The leucocyte count was 6,400. The temperature was normal.

*Read at the Sixteenth Annual Meeting of the American Association for Thoracic Surgery at Washington, D. C., May 9, 10, 11, 1933.

An x-ray picture, steroscopic, showed a large needle lying within the cardial shadow, deep in the chest nearer the bodies of the vertebrae than the sternum and inclined a little backward and downward, across the heart shadow. Under the fluoroscope the needle moved with the heartbeat, the left end more than the right. It seemed that it must be either in or behind the heart.

The electrocardiographic records were made and interpreted by Dr. W. W. Eakin and have been reported elsewhere. These notes are quoted from his records.

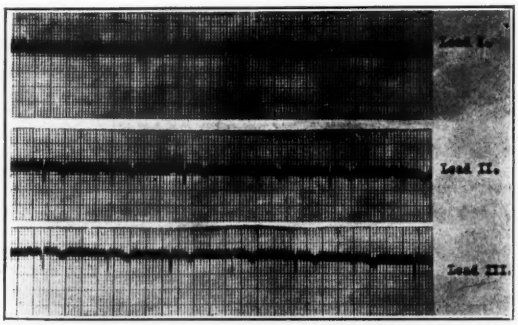

Fig. 1.—Tracing taken before operation. A low voltage of the QRS deflection and negative T deflections in the second and third leads may be observed.

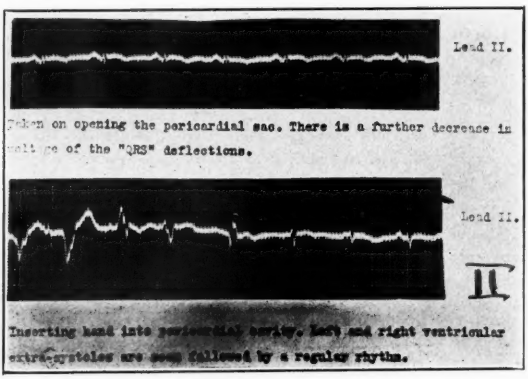

Fig. 2.

The electrocardiogram (Fig. 1) taken on admission shows a low voltage of the QRS deflections and an acutely negative T deflection in the second and third leads. This was taken as evidence of localized ventricular muscle damage. It resembles that seen in coronary thrombosis of some duration.

During her preoperative stay in the hospital her pain and disability increased notably until she was forced to lie on her back, afraid to move, as if the quickening of the heart rate on movement was associated with increased pain and respiratory distress.

The steady increase in pain and distress seemed to demand an attempt at removal, which was admittedly expected to be both difficult and dangerous. Here, as in all surgical difficulty, a free exposure was the first necessity. A review of the

different methods of approach to the heart showed, as is stated by Cutler,* that they fall into one of two general groups: (1) a midline incision which does not enter the pleura and (2) some form of lateral incision which does.

Through the midline incision alone can the heart be fully exposed. That it can be fully exposed through the midline is perhaps not so generally recognized as it should be. The midline approach, however, is easy, nearly bloodless, well under control, and is free from the respiratory complications of an open thorax. The most satisfactory method is known as the Duval-Barasty median thoracoabdominal pericardiotomy. It consists of a midline incision beginning at the level of the second rib and extending downward toward the umbilicus. The abdomen is opened in the midline up to the sternum. The diaphragm is separated from the sternum and the soft tissues are separated from its under surface. The sternum is then split up to the second rib (Cutler recommends that this be done with a motor driven circular saw but states that it may also be done with a pair of bone cutters). In this instance the splitting of the sternum was done quickly and easily with a mallet and chisel. This leaves a good clean bone edge which can be shaped away a little to

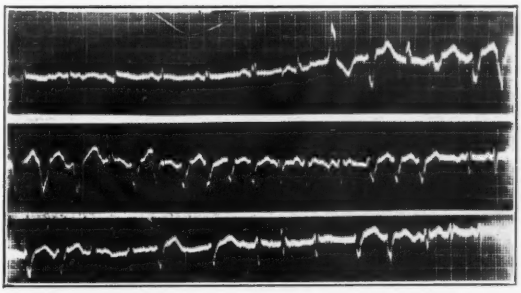

Fig. 3.—The tracing is a continuous one taken when the operator's hand was in contact with the needle. In the first part a change in form of the QRS deflections is seen followed by alternate right and left ventricular extrasystoles and a ventricular rhythm interrupted by an occasional supraventricular beat. In the last part a return to the normal is seen. The tracing is Lead II.

the right, and makes subsequent closure easy. The sternum was then cut toward the left, at the level of the second rib and the cartilage of the second rib was cut through with a knife. The sternum was then spread and as the pericardium appeared, the pleura was pushed away from its anterior surface with a gauze pad on forceps.

For full exposure the pericardium must be opened down to the diaphragm, the peritoneal contents packed away, and the diaphragm cut nearly to the crura. The incision of the diaphragm must work over a little to the left to avoid injury to the great veins. A very satisfactory exposure of the heart was obtained.

During the whole operation almost continuous electrocardiograph records were taken. Up to the opening of the pericardium no change was observed, but at this point there was a decrease in the voltage of the QRS and T deflections (Fig. 2).

On opening the pericardium a normal amount of clear fluid was found. The heart lay exposed, beating regularly with a distinct twist to the right with each ventricular beat. The muscle appeared normal and the coronary vessels were clearly seen. The pericardium was quite clean and smooth, except for a band of firm fibrous adhesions about three-fourths of an inch broad extending from the posterior surface of the left ventricle to the precardial wall. No sign of the needle could be found.

*Lewis: *Practice of Surgery.*

This manipulation brought out right and left ventricular extrasystoles followed by a normal rhythm, Fig. 3.

The needle not being free in the pericardium, the position of the single adhesion, in view of the position of the needle as revealed by the fluoroscope, suggested the hope that one end of the needle might be projecting from the heart at this point. The adhesion was therefore hooked up and cut but no needle was found. This manipulation was accompanied by a marked change in the electrocardiogram, with marked notching of the QRS deflection and one left ventricular extrasystole, followed by a return to the original (Fig. 4).

The heart was then taken in the right hand, the fingers feeling toward the base among the roots of the great vessels. After several such attempts, each of which

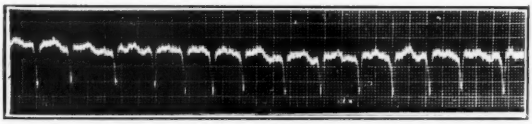

Fig. 4.—Ventricular tachycardia; rate 138 per minute. Taken when the operator was forcing the needle through the wall of the left ventricle. Lead II.

was stopped in a most irritating way by the anesthetist reporting that the patient had become white and the pulse had disappeared, a portion of the needle containing the eye was felt projecting from the posterior surface of the heart muscle in the region of the right auricle. Each time the needle was felt the patient turned white, the pulse disappeared, and the pupils either dilated or contracted. These manipulations were accompanied by changes in the electrocardiograph tracings, a prominent S-wave was replaced by a prominent and notched R deflection which was followed by right and left ventricular extrasystoles, and then a ventricular rhythm

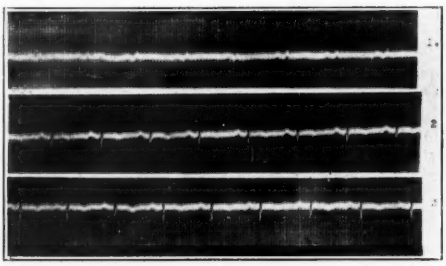

Fig. 5.—Taken just before leaving the operating table. Regular rhythm; rate 90 per minute. The voltage is somewhat lower than before operation.

interrupted by an occasional supraventricular beat. With release of the heart, color and pulse returned promptly each time. It was then recognized that with each attempt to feel the needle, the right thumb pressed on and occluded the right auricle, a procedure which is recommended for large wounds of the heart to control bleeding temporarily by blocking the entrance of the blood to the ventricle.

These maneuvers indicated: first, that the heart could tolerate compression of the auricle for five or six seconds and recover its rhythm; second, that the blunt end of the needle was the one felt and the direction was toward the adhesion on the posterior surface of the left ventricle; third, that the position and length of the needle made an attempt to grasp it too dangerous. It could only be felt through soft tissues except on one occasion, and any effort to get hold of it would

almost certainly have led to the tearing of either the auricle or the vein. The only way, it seemed, was to thrust the needle in the direction of its axis, through the heart, and make its point appear through the muscle of the left ventricle, near the adhesion which had been cut earlier in the operation. The hand was then placed about the heart, the fingers grasping the ventricle. The thumb sought for and found the projecting base of the needle and thrust it in the direction of its axis against the fingers, thus compressing the heart and making the point appear for about one-half inch almost at the old adhesion. At this point the pulse disappeared and the patient stopped breathing. The point of the needle was grasped between third and fourth fingers and drawn down to the left. As the thumb released its pressure on the auricle, the heart recovered its rhythm. There was extraordinarily little bleeding. The needle was 7 cm. long, the only thing which saved the situation.

During this part of the procedure the electrocardiogram showed signs of ventricular tachycardia. (Fig. 4.) During the compression of the heart there developed an irregular rhythm of right ventricular origin. This rhythm was irregular and suggested that it was not far removed from ventricular fibrillation. On release the heart returned to its original rhythm.

Fig. 6.—Picture taken November, 1932, five months after operation.

At no time did the pulsation of the heart cease or the muscle lose its tone.

After waiting a few minutes to be sure that the bleeding from the needle hole amounted to nothing and for the heart to recover its tone, the pericardium was closed, the peritoneum sutured, the sternum put into place, and the wound closed without drainage.

The patient left the table in good condition—pulse 90, blood pressure 120/70.

Electrocardiograph tracings taken just before leaving the operating room showed a regular rhythm of 90 per minute; the voltage was somewhat lower than before operation. (Fig. 5.)

Shortly afterward an attempt was made to reconstruct the position of the needle. This was done by placing it by feel in a heart before removal at autopsy. This suggests that the needle had pierced the heart from its auricle through the septum, crossed the left ventricle and nearly projected through the wall of the left ventricle at the point where the adhesion had formed. It suggested that the adhesion had formed as the result of the mechanical irritation of the point of the needle, which must have projected at some time.

The operation was performed on June 17, 1932. The following day the pulse averaged about 120 per minute, blood pressure 110/80. On June 21, the respirations

increased to 40 and she was distressed by pain. There was probably a small collection of fluid in the pericardial sac. The electrocardiogram showed a gradual return to a tracing not unlike that before operation. She left the hospital July 8, free from all symptoms.

July 20, 1932, she returned for an electrocardiogram, which showed that the T deflection was now upright in Leads I and II, but negative in Lead III. This was taken as evidence of recovery of the heart muscle. She was brought back again in November, 1932. She had regained full activity, the electrocardiogram had returned nearly to normal. It showed only a rather low voltage in the QRS.

MEDICAL ARTS BUILDING.

DISCUSSION

DR. T. C. DAVISON (ATLANTA).—At our last meeting in Washington I reported a case on the removal of a 25 calibre bullet from the pericardium, and gave the reaction of the heart before and during the operation, but unfortunately, no electrocardiogram was made. This operation was performed under local anesthesia, and the actions of the heart were recorded as it was held in the operator's hand. I explained the manipulations as the bullet was removed from a small abscess in the pericardium. The operation was successful.

Department of Reviews and Abstracts

CONDUCTED BY DUFF S. ALLEN, M.D., ASSOCIATE EDITOR

Selected Abstracts

Experimental Pneumococcus Lobar Pneumonia in the Dog.

I. Method of Production.

E. E. Terrell, O. H. Robertson, and L. T. Coggeshall.

II. Pathology.

O. H. Robertson, L. T. Coggeshall and E. E. Terrell.

III. Pathogenesis.

O. H. Robertson, L. T. Coggeshall, and E. E. Terrell. Jour. Clin. Invest. **12**: 393-493, March, 1933.

Pneumococcic lobar pneumonia was produced in dogs by the instillation of from 0.5-0.0001 c.c. of a starch-broth mixture to which a virulent culture of pneumococcus Type I had been added, into the terminal bronchi through a long radio-opaque catheter. The placing of the catheter in the lung was generally carried out under the fluoroscope.

Animals so treated in almost every instance developed a typical lobar consolidation. Those animals receiving the larger doses invariably died, while animals receiving a smaller infecting dose ran a course which simulated rather closely lobar pneumonia in man, with subsequent resolution and recovery.

Pathology:

In general, three types of lesions were observed: (1) In this group the pneumonic process tended to remain localized to the lungs, generally to one or two lobes and blood stream infection occurred in only a few animals. The picture both macroscopically and microscopically resembled closely that seen in man, although the stage of grey hepatization was never as pronounced as is generally observed in the latter. Resolution was individualized by the "macrophage reaction" a phenomenon characterized by the desquamation of the swollen septal cells into the alveolar spaces, where they became actively phagocytic. (2) In the animals falling within this class there was blood stream invasion in most cases and a rapidly progressing pneu-

monia, with complete consolidation in the initially infected lobes, and a scattered type of consolidation with marked pulmonary edema in the other lobes to which the process had advanced. In these lungs the cellular exudate was less uniform, the number of red cells in the exudate much greater, and the lung architecture less well preserved.

(3) Here the infection was generalized and much more severe and the animals generally died in two to three days with massive blood stream invasion and the pneumonic process more closely resembled a lobular pneumonia.

It was felt that the process in dog closely resembled that seen in man. In general the lesion failed to be everywhere as uniform in the experimental animal as in the human. The spread was as a rule more rapid and resolution generally set in before the stage of grey hepatization became markedly evident. However, the course of the disease in the dogs, with resolution and recovery and the presence of more devastating types of the disease simulate closely human lobar pneumonia.

Pathogenesis:

The authors have traced the process through the various disease stages, especially those early stages not seen in man. At one hour the picture was that of an early acute infection, there was hemorrhage, edema, cellular infiltration and the perivascular edema was marked; the pneumococci were spreading outward from the main lesion into the normal surrounding lung. At three hours the lesion appeared essentially the same as at one hour except for a larger area of involvement. There the pneumococci were found at great distances from the original lesion in alveolae which otherwise appeared normal.

In the early time periods the bacteria wandered far out into the normal lung tissue, where they were seen lying free in the alveoli and near the alveolar wall, some even appearing to have entered the septa. Wherever the organisms had advanced, their wake was followed by a pronounced edema which filled most of the alveoli and caused marked perivascular swelling. In those periods where the edema was great (six-hour period) the pneumococci were less readily seen and it was not until the twelve-hour lesion was studied that they were again seen in great numbers.

At the six-hour period the consolidation lagged far behind the advancing zone of edema, although in the area of the initial infection the picture was essentially that of an early lobar polymorphonuclear leucocytes and red blood cells and the alveolar walls were outlined.

At twelve hours consolidation was more marked than at six hours and in the animals which had received large infecting doses the whole lobe was in some instances consolidated. The number of organisms seen in the lung sections was much greater.

Study of the twenty-four-hour lesion revealed that the initially infected lobe was almost completely consolidated with the exception of an area 1-2 cm. from the hilus on the posterior lobe surface. The pleural surfaces were covered with fibrin but the pleural cavity was dry. Microscopically the process was much more uniform than seen before, the alveoli were packed with cells and only occasional alveoli filled with edema fluid alone were seen. Lacunae which had been present in the consolidated portions of the early lesions were not present. In the older portions of the lesions the macrophage reaction characteristic of resolution was present.

Wm. M. Tuttle.

Pol. N. Coryllos. The Importance of Atelectasis in Pulmonary Tuberculosis. Am. Rev. Tuberculosis **28**: 1-26, July, 1933.

The author discusses the rôle played by bronchial occlusion in the formation and adequate collapse of tuberculous cavities and the relation of tissue anoxemia in the atelectatic lung to the formation of fibrous tissue.

The tuberculous process starts as a parenchymatous infiltration, which sooner or later caseates and by the establishment of an adequate bronchial communication with the caseous area, the softened material is coughed up leaving a cavity. This cavity irrespective of the outline of the caseous area, becomes with the expulsion of the caseous material, spherical in shape, due to a pressure of one atmosphere within the cavity and a negative pressure of –5 mm. Hg in the intrapleural space. Thus the cavity is distended and kept so by the same physiologic process which preserves the lungs in a state of relative expansion; namely, a greater pressure within the cavity working against a lesser pressure on the outside. It is obvious, therefore, that tuberculous pulmonary cavities will remain uncollapsed as long as this pressure relation is maintained. It is only after spontaneous or artificial pneumothorax or some of the various surgical collapse procedures have increased the intrapleural pressure and when by kinking or other means the bronchial communication of the cavity is stenosed and the contained air absorbed that the cavity collapses due to reversed pressure conditions. Adhesions holding the lung tissue over the cavity to the chest wall, prevent collapse of the cavity, not by acting as ropes to hold the walls out, but by preventing compression of the smaller bronchi in the surrounding lung tissue and thus a closure of the air supply to the cavity.

Spontaneous closure of cavities in a surprisingly short time in the absence of therapeutic measures to alter the intrapleural pressure is the result of stenosis of the communicating bronchi due to tuberculous involvement of these passages.

Fibrous tissue formation in tuberculosis may be due to the fact that the disease is one of the connective tissue element, but it may also be due to the avascularity of tuberculous lesions; the resulting tissue anoxemia stimulating a pronounced fibroblastic response. Similarly atelectasis by reducing the circulation and giving rise to an anoxemia state, may thus stimulate fibrous tissue formation in the collapsed lung.

The disappearance of the acid fast organisms from the collapsed lung is the result of this bacteria's inability to live in an atmosphere of low oxygen tension. (Richardson, Loebel, and Shorr.)

Wm. M. Tuttle.

Badham, Charles: Coal Miners Lung. M. J. Australia. **1:** 511, 1933.

This investigation is a chemical and pathologic study of the lungs from 31 persons. Nineteen of these were coal miners, 8 had worked in metal mines, 3 were sandstone masons, 1 was a metal polisher and 1 an ore miller, and 2 had died from tuberculosis which had no possible occupational etiology.

The lesion in the lung of the Welsh coal miner was usually an upper lobe consolidation with nontuberculous cavitation, and there was often an extension of this primary consolidation to form a cuirass over the upper parts of the lung.

Carbon retains silica and silicates, so that it is very difficult to extract silica from coal dust with sodium hydroxide. Badham suspects that this bond between silica and carbon plays a part in their insolubility in the body fluids.

The petrologic microscope was found unsuitable for use in the examination for quartz in these lungs, since most of the particles were less than 3 microns in diameter. The method of analysis chosen, therefore, was to cut 200 to 300 gm. of lung into small pieces, dry, pulverize and heat until coked, grind again and heat. This drives off all the carbon. The ash was then analyzed for silica and silicates. Badham finds that there are several different types of pulmonary fibrosis in coal miners (in New South Wales) according to whether the patient had worked in other dusts. In certain of these he found silicatosis due to the silicates and not to quartz or silica. The evidence is opposed to the probability that tuberculosis can be aggravated or caused by coal dust with a high carbonaceous content. The combination of carbon and silicates produces a characteristic lesion.

The x-ray picture alone is insufficient for the diagnosis of silicosis or pneumoconiosis. The complication of tuberculosis cannot be diagnosed from the x-ray picture. Tubercle bacilli in the sputum must be found to make the diagnosis.

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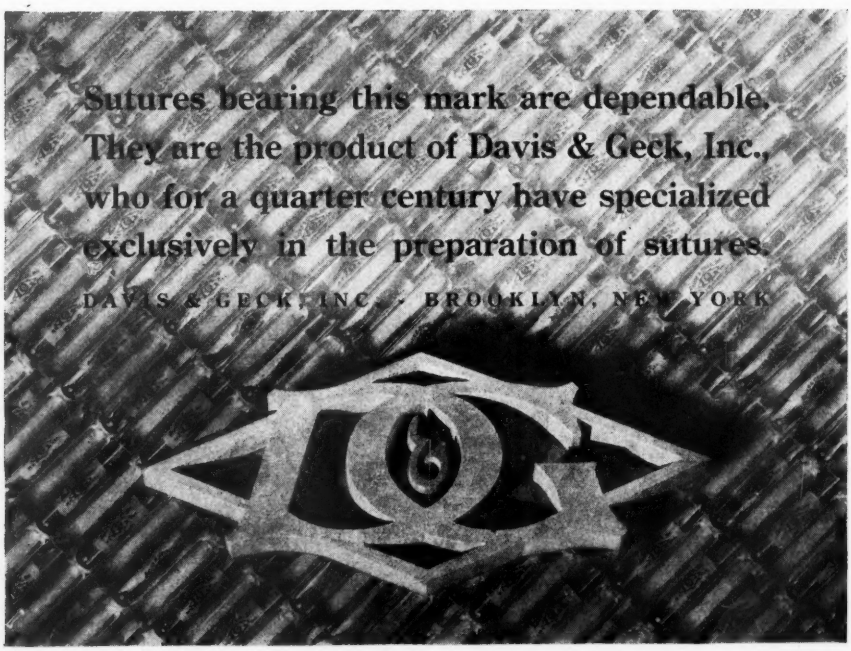
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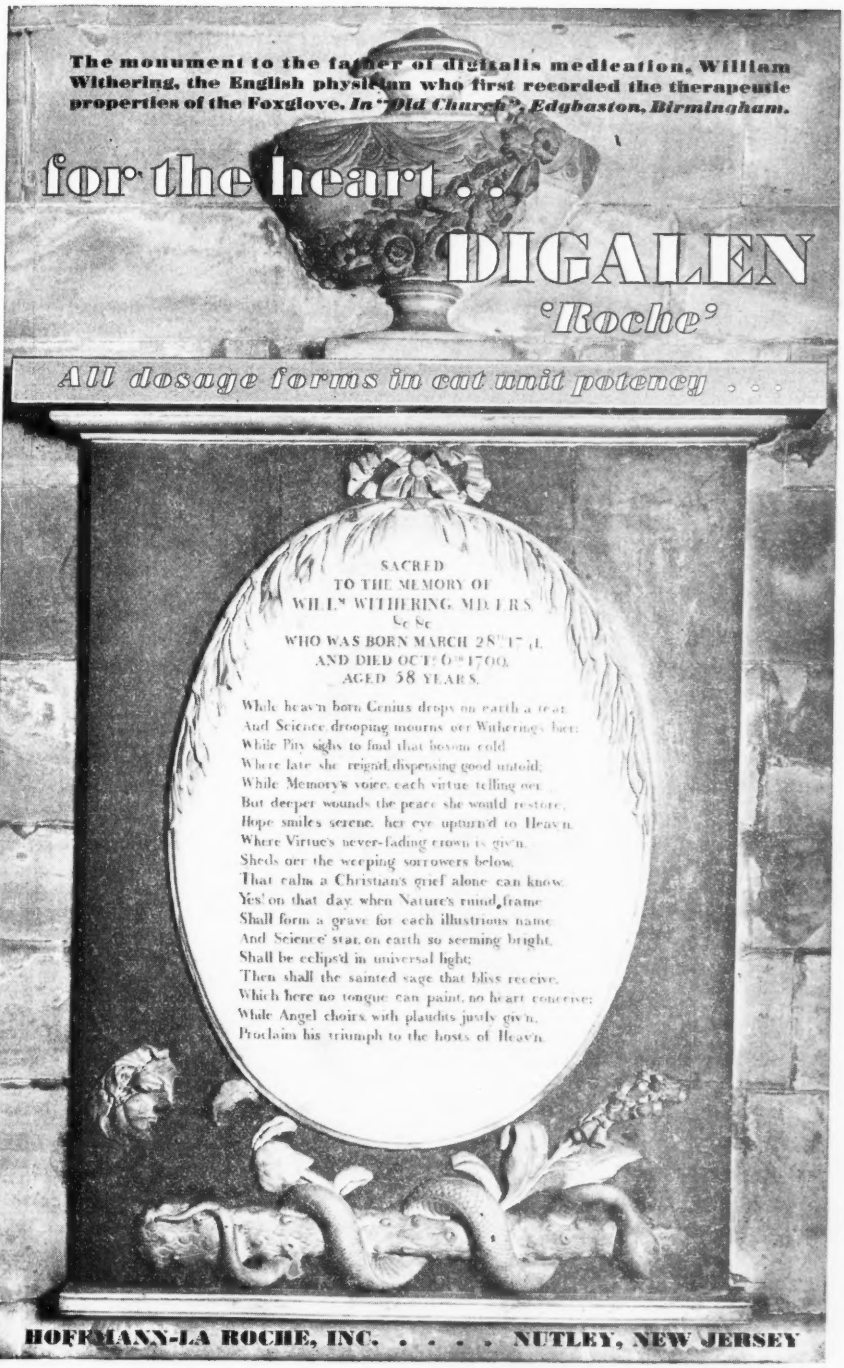
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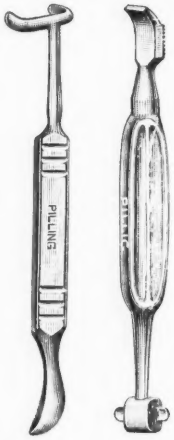

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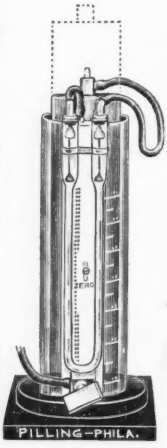

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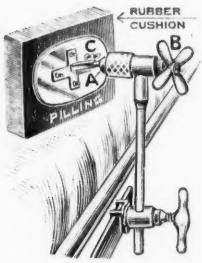

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